IN THE NAME OF GOD

ARRYTHMIAS

Dr.Sima Sayah

TYPES OF BYPASS TRACTS

Bypass tracts (BTs) are remnants of the atrioventricular (AV) connections caused by incomplete embryological development of the AV annulus and failure of the fibrous separation between the atria and ventricles.

THERE ARE SEVERAL TYPES OF BTS

1.AV junction bypassing the atrioventricular node–His-Purkinje system (AVN-HPS).

Atrionodal BTs connect the atrium to the distal or compact AVN

Atrio-Hisian BTs connect the atrium to the His bundle (HB)

Atriofascicular pathways

Nodofascicular pathways,

HB (fasciculoventricular)

TYPES OF PREEXCITATION SYNDROMES

The term *syndrome* is used when the anatomical variant is responsible for tachycardia

In the Wolff-Parkinson-White (WPW)syndrome, AV conduction occurs, partiallyor entirely, through an AV BT, which

results in earlier activation (preexcitation)

of the ventricles than if the impulse had

traveled through the AVN.

In the case of Lown-Ganong-Levine (LGL) syndrome,preexcitation purportedly occurs via

atrio-Hisian BTs or, alternatively, no BT is

present and enhanced AVN conduction

accounts for the ECG findings.

The net effect is a short PR interval without delta wave or QRS prolongation

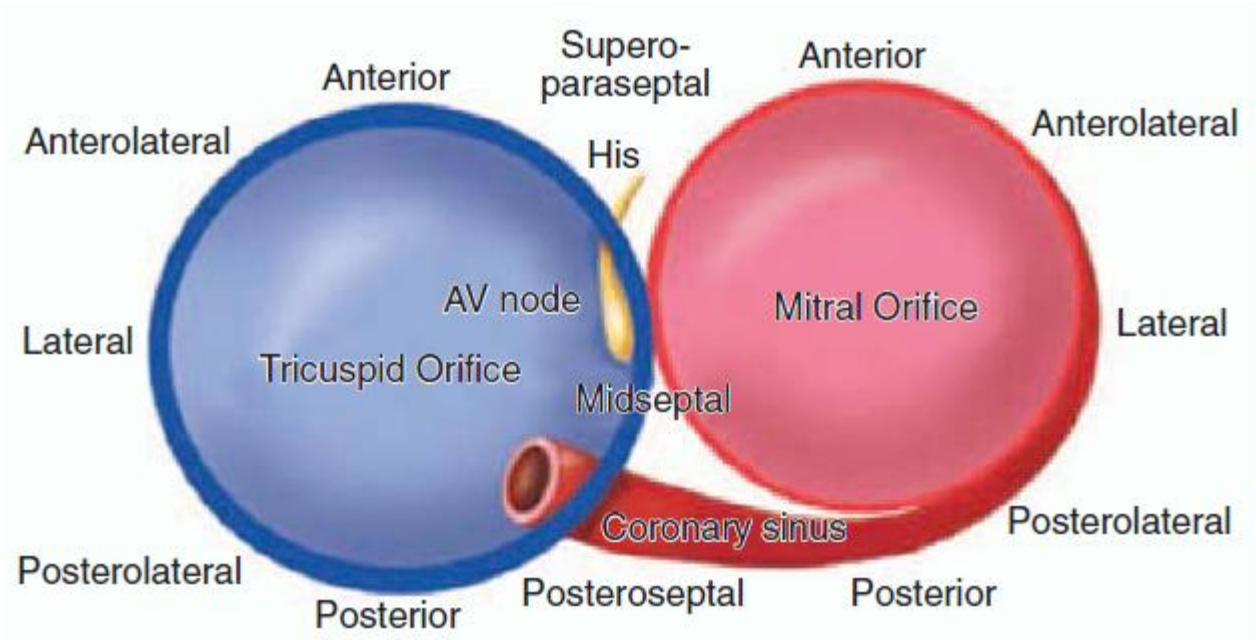
The Mahaim variant of preexcitation does not typically result in a delta wave, because these pathways, which usually terminate in the conducting system or in the ventricular myocardium close to the conducting system, conduct slowly and the AVN-HPS has adequate time to activate the ventricle predominantly.

Concealed AV BTs refer to AV BTs that do not manifest anterograde conduction and therefore do not result in ventricular preexcitation

Because they do not result in alteration of the QRS complex in the ECG, they cannot be detected by inspection of the surface ECG; they are called concealed.However, the concealed BT can conduct in a retrograde fashion

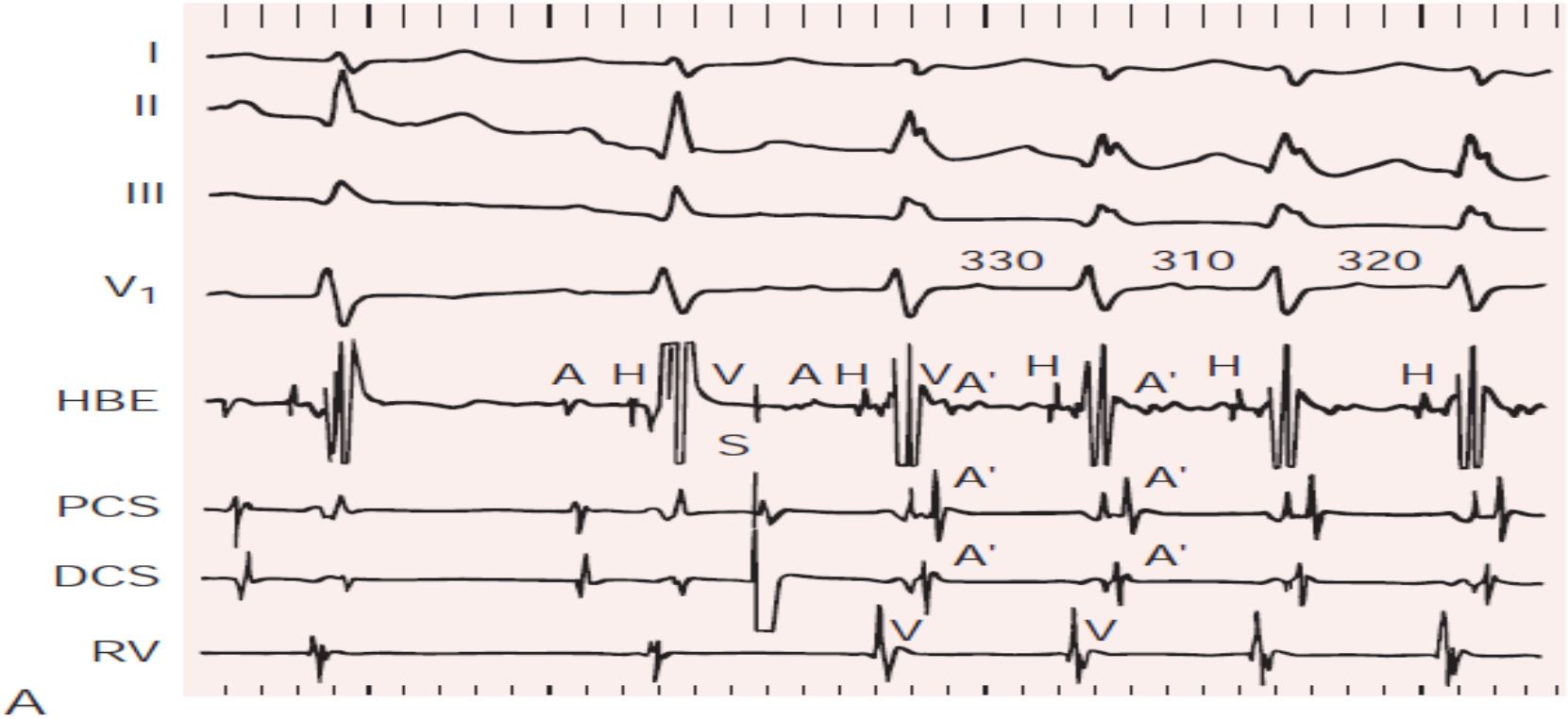
PATHOPHYSIOLOGY

1. Because the AV BT typically conducts faster than the AVN, the onset of ventricular activation is earlier than if depolarization occurred only via the AVN, resulting in a shortened PR (P-delta) interval.
2. because the BT exhibits nondecremental conduction, the early activation (P-delta interval) remains constant at allheart rates.


Anterior
Supero-
paraseptal
Anterior
Anterolateral
Anterolateral
His
AV node
Mitral Orifice
Lateral
Lateral
Tricuspid Orifice
Midseptal
Coronary sinus
Posterolateral
Posterolateral
Posterior
Posteroseptal
Posterior

REENTRY OVER A CONCEALED (RETROGRADE-ONLY) ACCESSORY PATHWAY

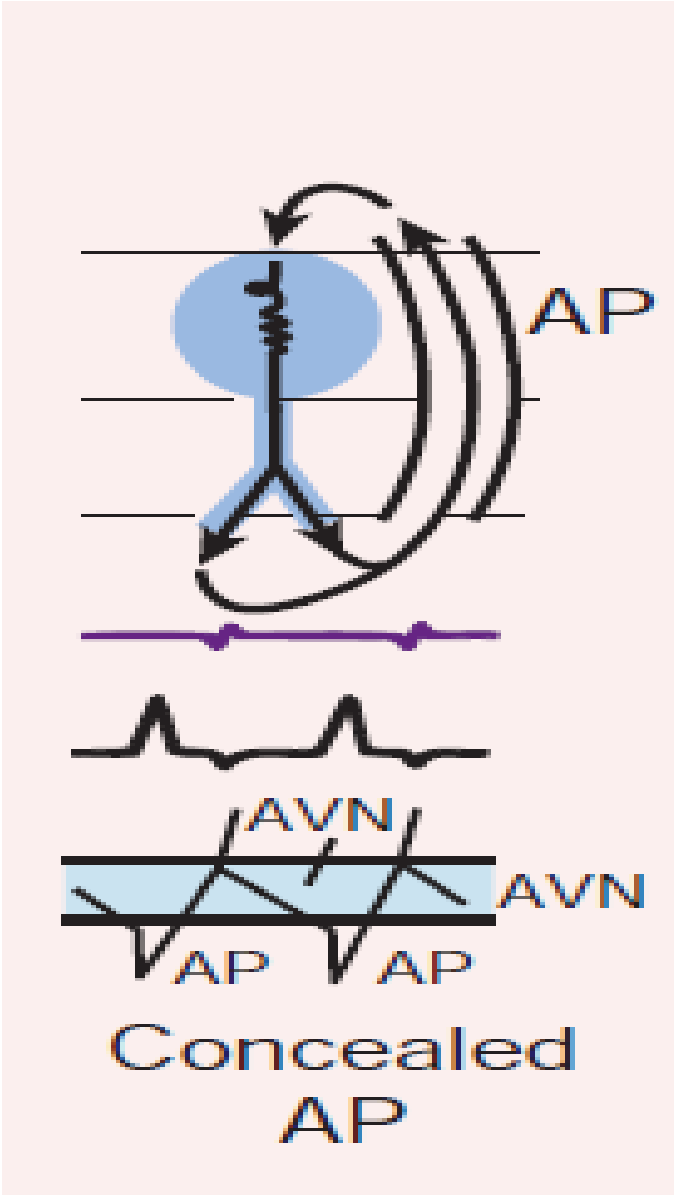
Electrocardiographic manifestations of WPW syndrome are absent



A premature stimulus in .the coronary sinus (S) precipitates SVT at a cycle length of approximately 330 milliseconds. The retrograde atrial activation sequence begins first in the distal coronary sinus (A′, DCS), followed by activation recorded in the proximal coronary sinus (PCS), low right atrium (HBE), and then the high right atrium (not shown). The QRS complex is normal and identical to the sinus-initiated QRS complex. (The terminal portion is slightly deformed by superimposition of the retrograde atrial recording.) Note that the RP interval is short and the PR interval is long. The shortest VA interval exceeds 65 milliseconds, consistent with conduction over a retrogradely conducting AV pathway.

B

Premature ventricular stimulation at a time when the His bundle is still refractory from anterograde activation during tachycardia shortens the A-A interval from 330 to 305 milliseconds without a change in the retrograde atrial activation sequence. (Note that no change occurs in the H-H interval when the RV stimulus [S] is delivered. H-H intervals are in msec in the HBE lead.) Thus the ventricular stimulus, despite His bundle refractoriness, still reaches the atrium and produces an identical retrograde atrial activation sequence. The only way that this finding can be explained is by conduction over a retrogradely conducting accessory pathway. Therefore the patient has a concealed accessory pathway with WPW syndrome. HRA = high right atrium; RV = right ventricle



Concealed AP

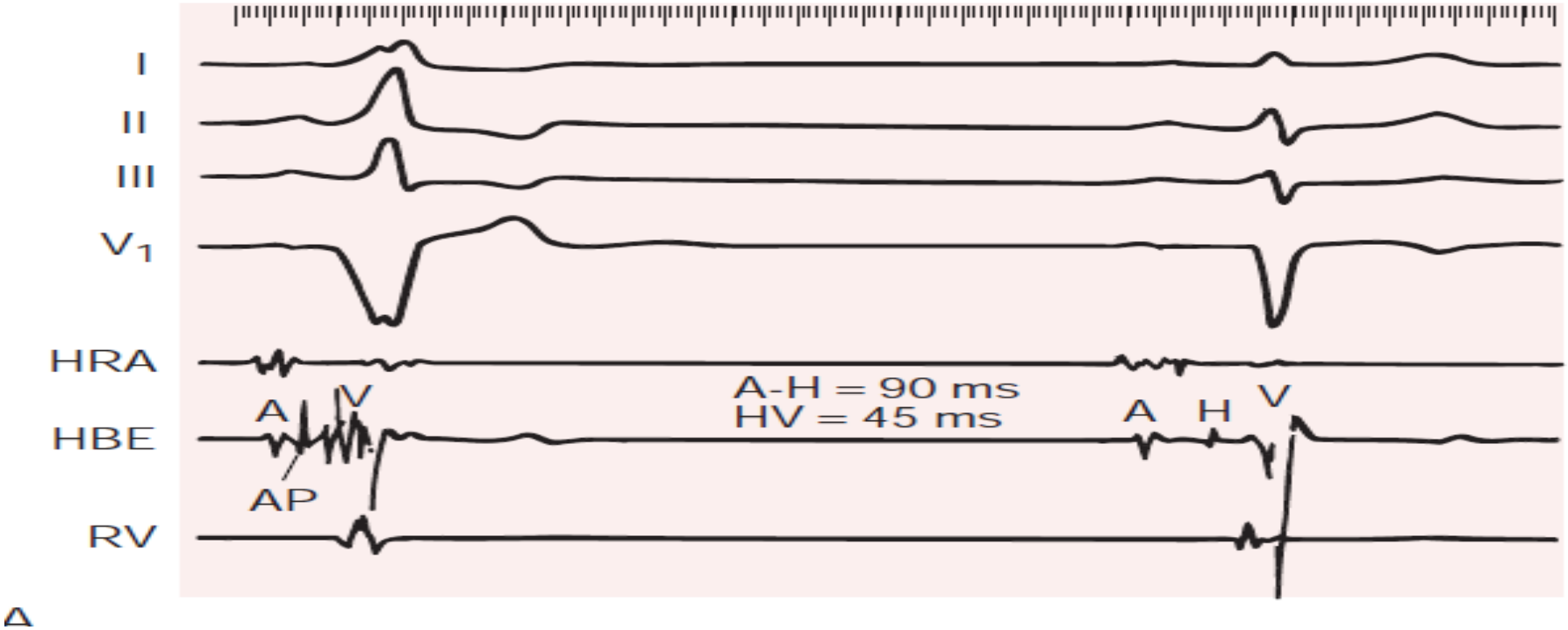
ELECTROCARDIOGRAPHIC EXAMINATION

the QRS complex is normal and the retrograde P wave occurs after completion of the QRS complex, in the ST segment, or early in the Twave

Sometimes the P wave is not clearly visible and can result in depression of the ST segment;

- retrograde P wave must occur after ventricular excitation,in contrast to AV nodal reentry, in which the atria are usually excited during ventricular activation
- The contour of the retrograde P wave can differ from that of the usual retrograde P wave because the atria may be activated eccentrically, that is, in a manner other than the normal retrograde activation sequence, which starts at the low right atrial septum as in AV nodal reentry.
- This eccentric activation occurs because the concealed accessory pathway in most cases is left sided (i.e., inserts into the left atrium), which makes the left atrium the first site of retrograde atrial activation and causes the retrograde P wave to be negative in lead I

because the tachycardia circuit involves the ventricles, if a functional bundle branch block occurs in the same ventricle in which the accessory pathway is located, the VA interval and cycle length of the tachycardia can become longer

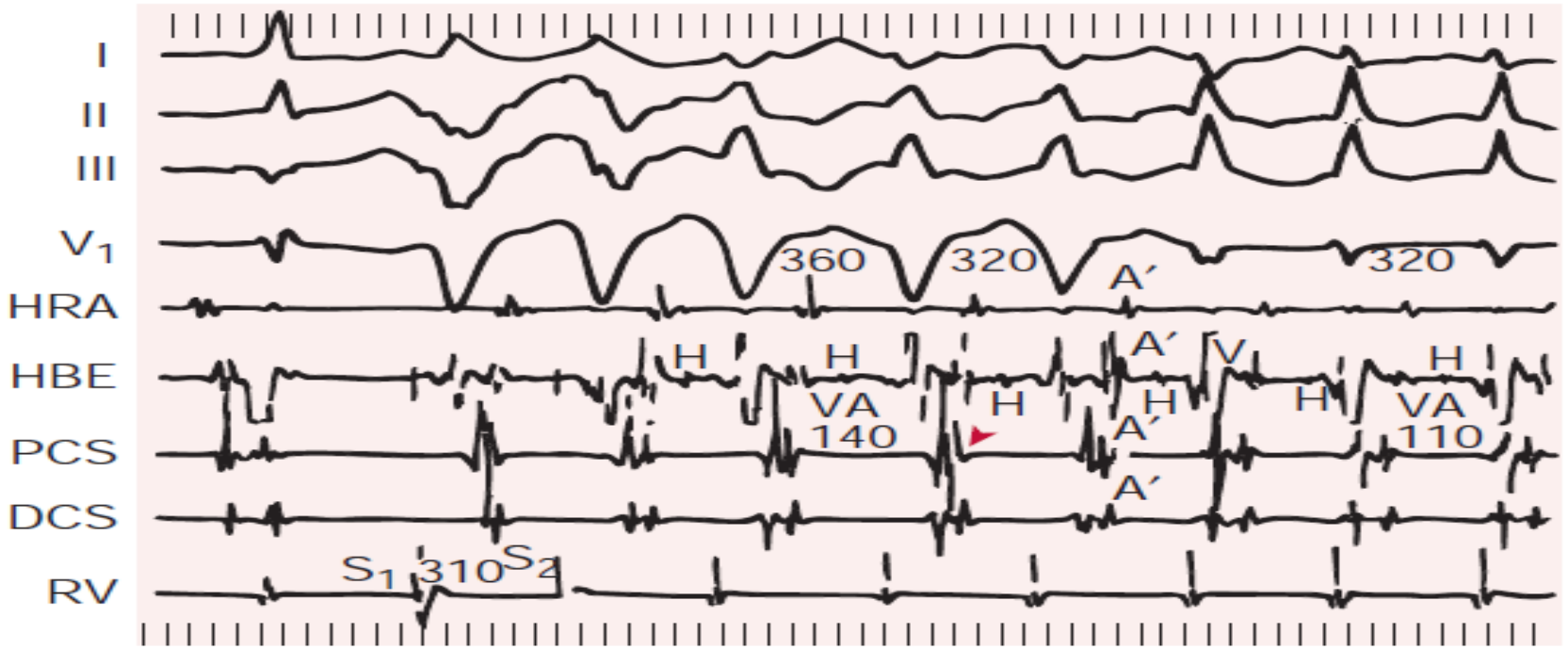


A

Recording of depolarization of an accessory pathway (AP) with a catheter electrode. The first QRS complex illustrates conduction over the AP. On the

scalar ECG, a short PR interval and delta wave (best seen in leads I and V1) are apparent. His-bundle activation is buried within the ventricular complex. In the following complex, conduction has been blocked over the AP, and a normal QRS complex results. His-bundle activation clearly precedes the onset of ventricular depolarization by 45 milliseconds.

The A-H interval for this complex is 90 milliseconds



Influence of a functional ipsilateral bundle branch block on the VA interval during AV reciprocating tachycardia. Partial preexcitation can be noted in the sinus-initiated complex (first complex). Two premature ventricular stimuli (S1, S2) initiate a sustained SVT that persists with a left bundle branch

block for several complexes before finally reverting to normal. The retrograde atrial activation sequence is recorded first in the proximal coronary sinus lead (*arrowhead,* PCS),

then in the distal coronary sinus lead (DCS) and low right atrium (HBE), and then high in the right atrium (HRA). During the functional bundle branch block, the VA interval in the

PCS lead is 140 milliseconds, which shortens to 110 milliseconds when the QRS complex reverts to normal. Such behavior is characteristic of a left-sided accessory pathway with

prolongation of the reentrant pathway by the functional left bundle branch block

EXAMPLE, THE TACHYCARDIA CIRCUIT TRAVELS FROM THE ATRIUM > THE AV NODE–HIS BUNDLE, > THE RIGHT VENTRICLE, > THE SEPTUM, > THE LEFT VENTRICLE, > THE ACCESSORY PATHWAY, AND THEN BACK TO THE ATRIUM.

- The additional time required for the impulse to travel across the septum from the right to the left ventricle before reaching the accessory pathway and atrium lengthens the VA interval, which consequently lengthens the cycle of the tachycardia by an equal amount, assuming that no other changes in conduction times occur within the circuit.

THE PRESENCE OF AN IPSILATERAL BUNDLE BRANCH BLOCK CAN FACILITATE REENTRY AND CAUSE AN INCESSANT AV REENTRANT TACHYCARDIA.

lengthening of the tachycardia cycle by

more than 30 milliseconds during an ipsilateral functional bundle branch block is diagnostic of a free wall accessory pathway if the lengthening can be shown to be caused by VA prolongation only and not by prolongation of the H-V interval (which can develop with the appearance of a bundle branch blo

A functional bundle branch block in the ventricle contralateral to the accessory pathway does not lengthen the tachycardia cycle if the H-V interval does not lengthen.

SEPTAL ACCESSORY PATHWAY

1. retrograde atrial activation is normal (concentric) because it occurs retrogradely up the septum

2.the VA interval and cycle length of the tachycardia increase 25 milliseconds or less with the development of an ipsilateral functional bundle branch block..

CLINICAL FEATURES

Concealed accessory pathways are estimated to be present in approximately 30% of patients with apparent SVT referred for EP evaluation.

Most of these accessory pathways are located between the left ventricle and left atrium or in the posteroseptal area, less commonly between the right ventricle and right atrium.

Tachycardia rates tend to be somewhat faster than those occurring in AV nodal reentry (200 beats/min)

Syncope can occur because the rapid ventricular rate fails to

provide adequate cerebral circulation or because the tachyarrhythmia

depresses the sinus pacemaker and causes a period of asystole

when the tachyarrhythmia terminates

MANAGEMENT

The therapeutic approach to termination of this form of tachycardia acutely is as outlined for AV nodal reentry because the AV node is a critical part of the circuit here as well

In general, the most successful method is to produce a transient AV nodal block; therefore, vagal maneuvers and intravenous administration of adenosine, verapamil, or diltiazem and beta blockers are acceptable choices

RF CATHETER ABLATION IS CURATIVE, HAS LOW RISK, AND SHOULD BE CONSIDERED EARLY FOR SYMPTOMATIC PATIENTS

RF catheter ablation and antiarrhythmic

agents that prolong the activation time or refractory period

in the accessory pathway need to be considered for chronic prophylactic

therapy,

PREEXCITATION SYNDROME
ELECTROCARDIOGRAPHIC RECOGNITION

Three basic features typify the electrocardiographic abnormalities

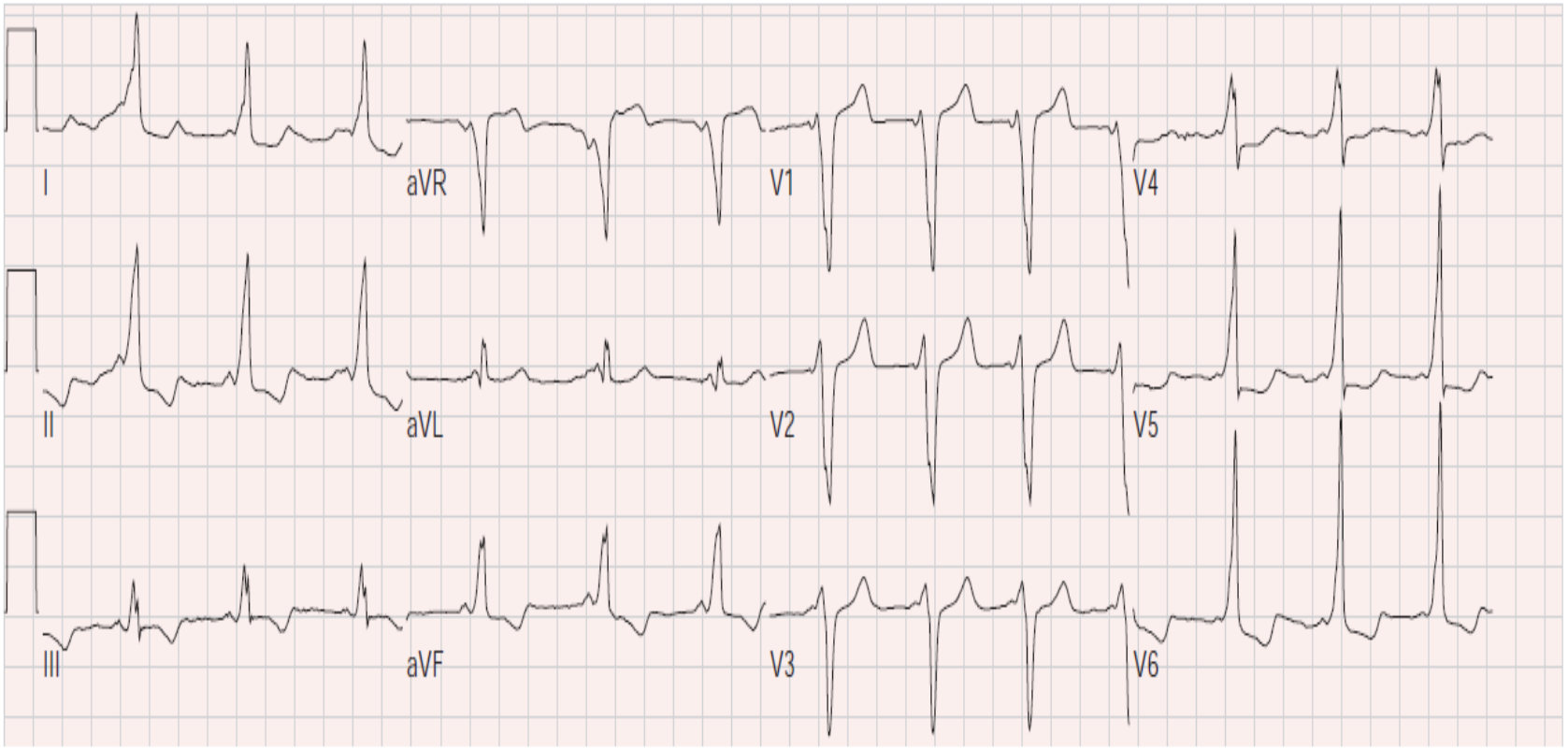
in patients with the usual form of WPW conduction caused

by an AV connection:

(1) PR interval less than 120 milliseconds during sinus rhythm; (2) QRS complex duration exceeding 120 milliseconds

with a slurred, slowly rising onset of the QRS in some leads

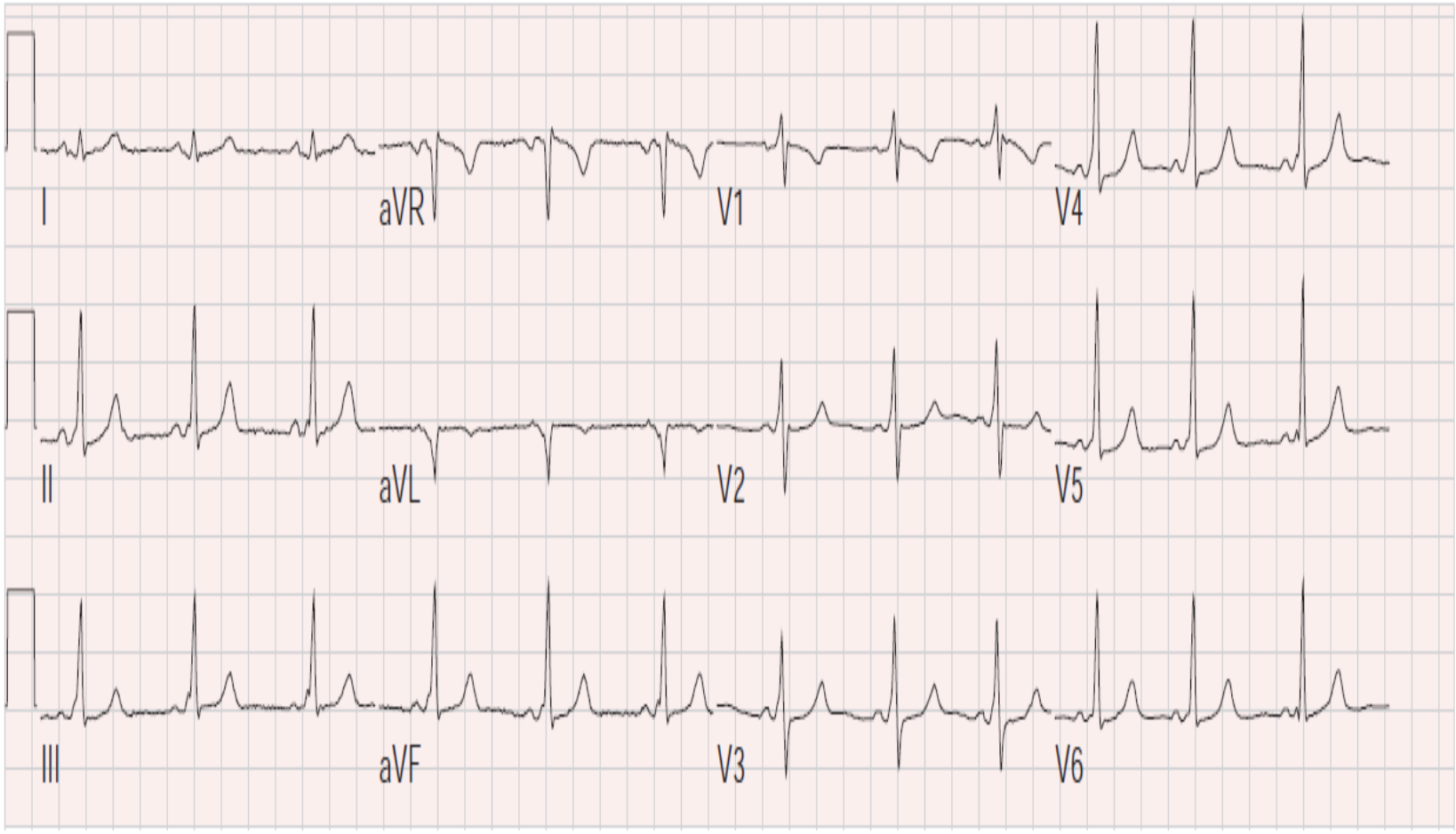
(delta wave) and usually a normal terminal QRS portion; and (3)secondary ST-T wave changes that are generally directed in an opposite direction to the major delta and QRS vectors.

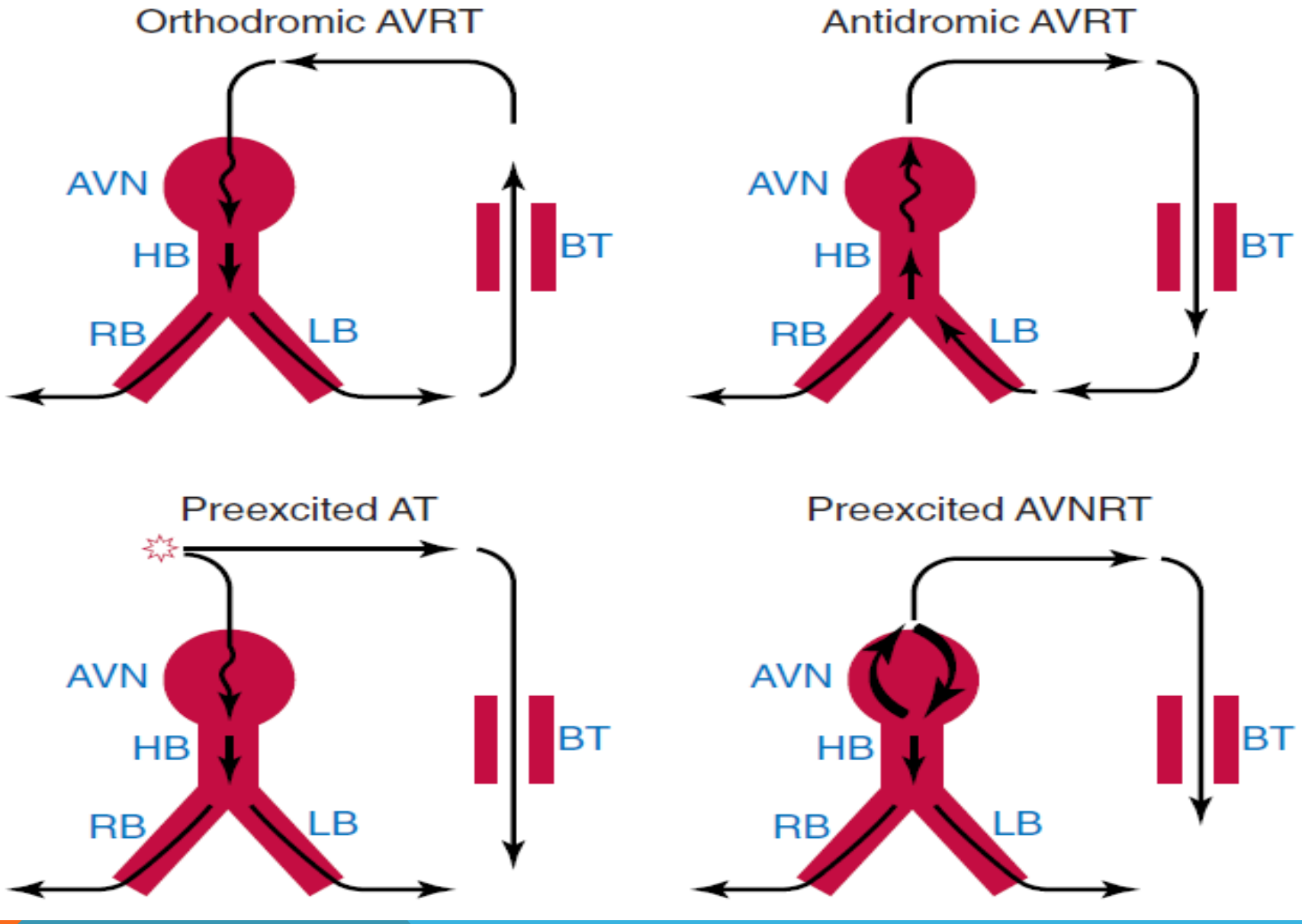


Right anteroseptal accessory pathway

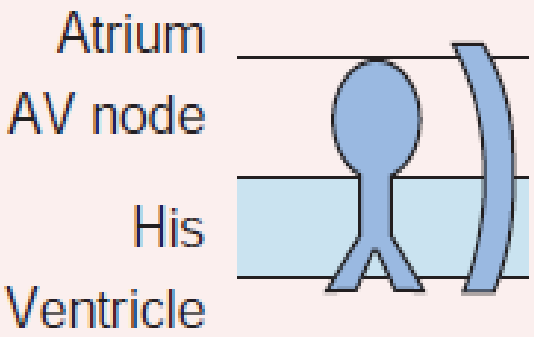
RIGHT POSTEROSEPTAL ACCESSORY PATHWAY ATRIAL FIBRILLATION IS PRESENT.

LEFT LATERAL ACCESSORY PATHWAY



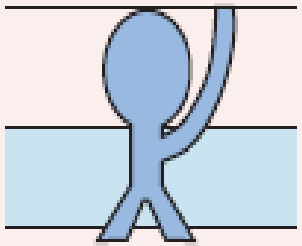

Orthodromic AVRT
AVN
HB
RB
LB
BT
Antidromic AVRT
AVN
HB
RB
LB
BT
Preexcited AT
AVN
HB
RB
LB
BT
Preexcited AVNRT
AVN
HB
RB
LB
BT

A



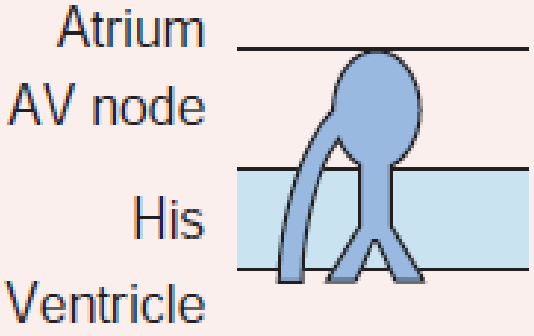
Atrioventricular

B



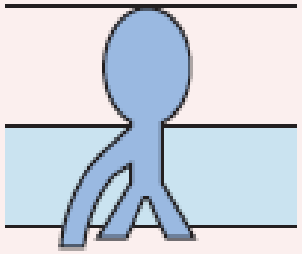
Atriohisian

C



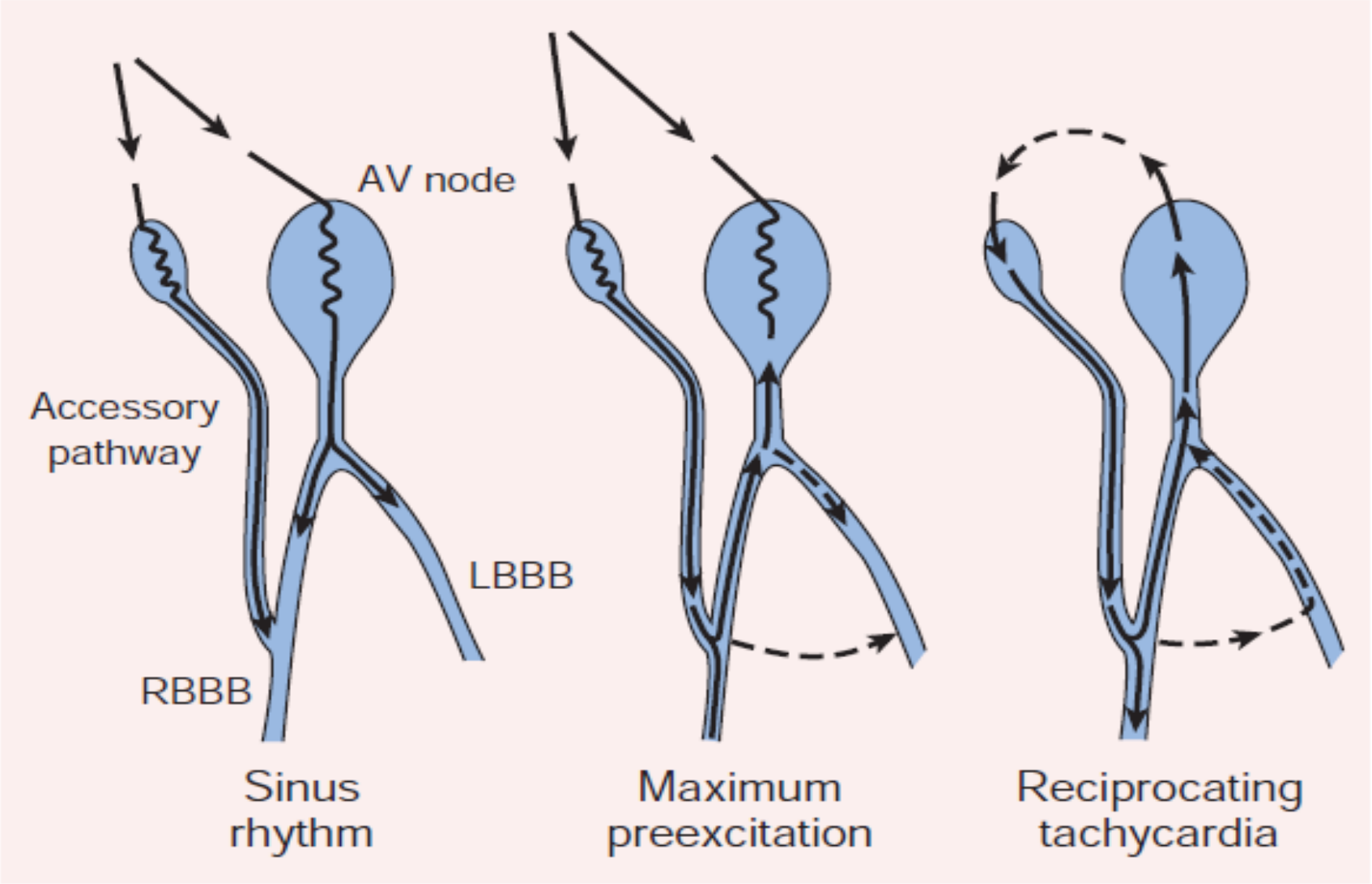
Nodoventricular

D



Fasciculoventricular

CURRENT CONCEPT OF THE NODOFASCICULAR ACCESSORY PATHWAY, IN WHICH THE ACCESSORY PATHWAY IS AN AV COMMUNICATION WITH AV NODE–LIKE PROPERTIES.
SINUS RHYTHM RESULTS IN A FUSION QRS COMPLEX, AS IN THE USUAL FORM OF WPW SYNDROME



E

No preexcitation is generally apparent during sinus rhythm, but it can be exposed by premature right atrial stimulation

The usual absence of retrograde conduction in these pathways produces only an antidromic AV reentry tachycardia ("preexcited" tachycardia) characterized

by anterograde conduction over the accessory pathway

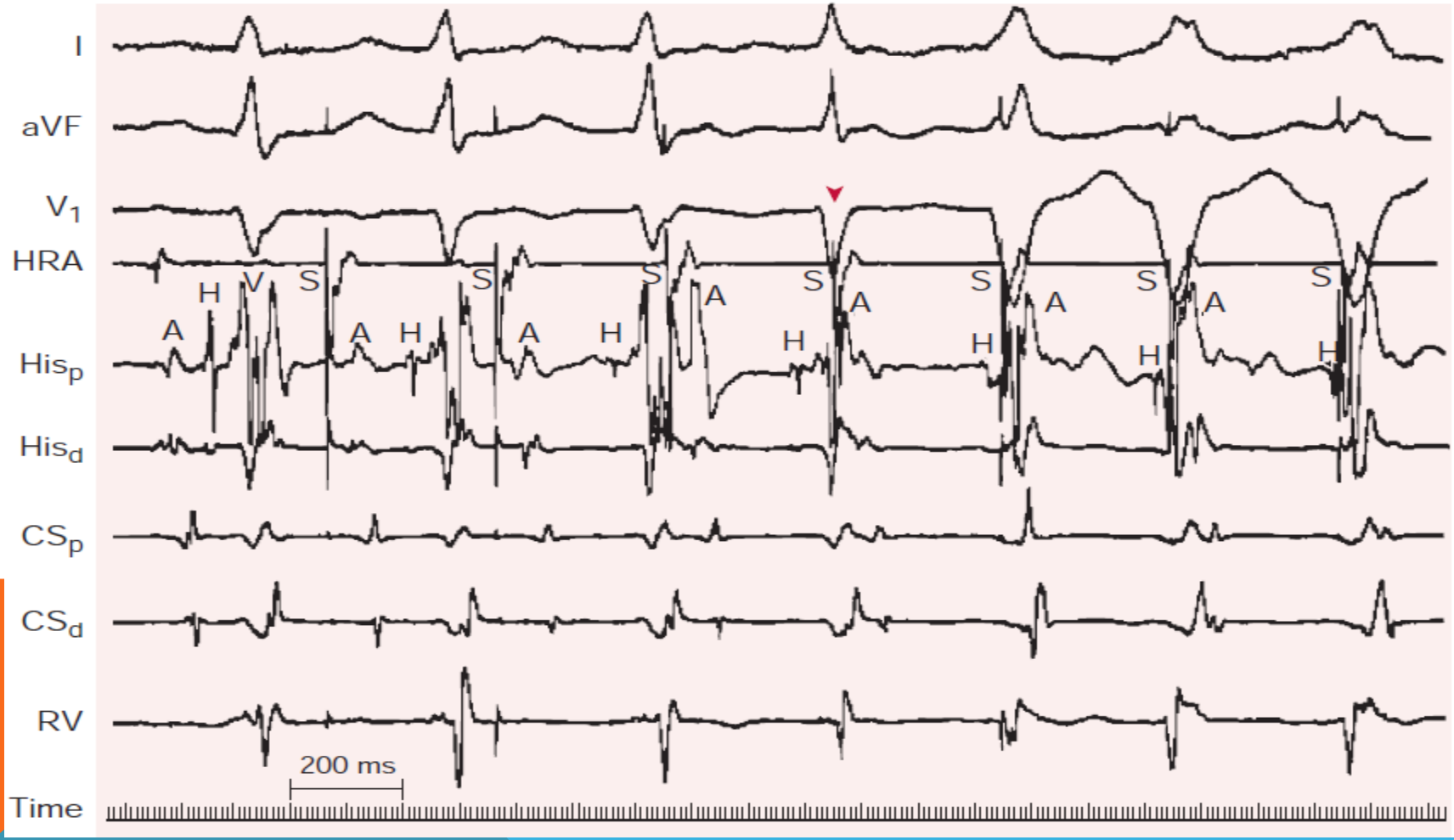
and retrograde conduction over the right bundle branch–His bundle–AV node, thus making the atrium a necessary part of the circuit.

THE PREEXCITED TACHYCARDIA HAS A LEFT BUNDLE BRANCH BLOCK PATTERN, LONG
AV INTERVAL (BECAUSE OF THE LONG CONDUCTION TIME OVER THE ACCESSORY
PATHWAY), AND SHORT VA INTERVAL.

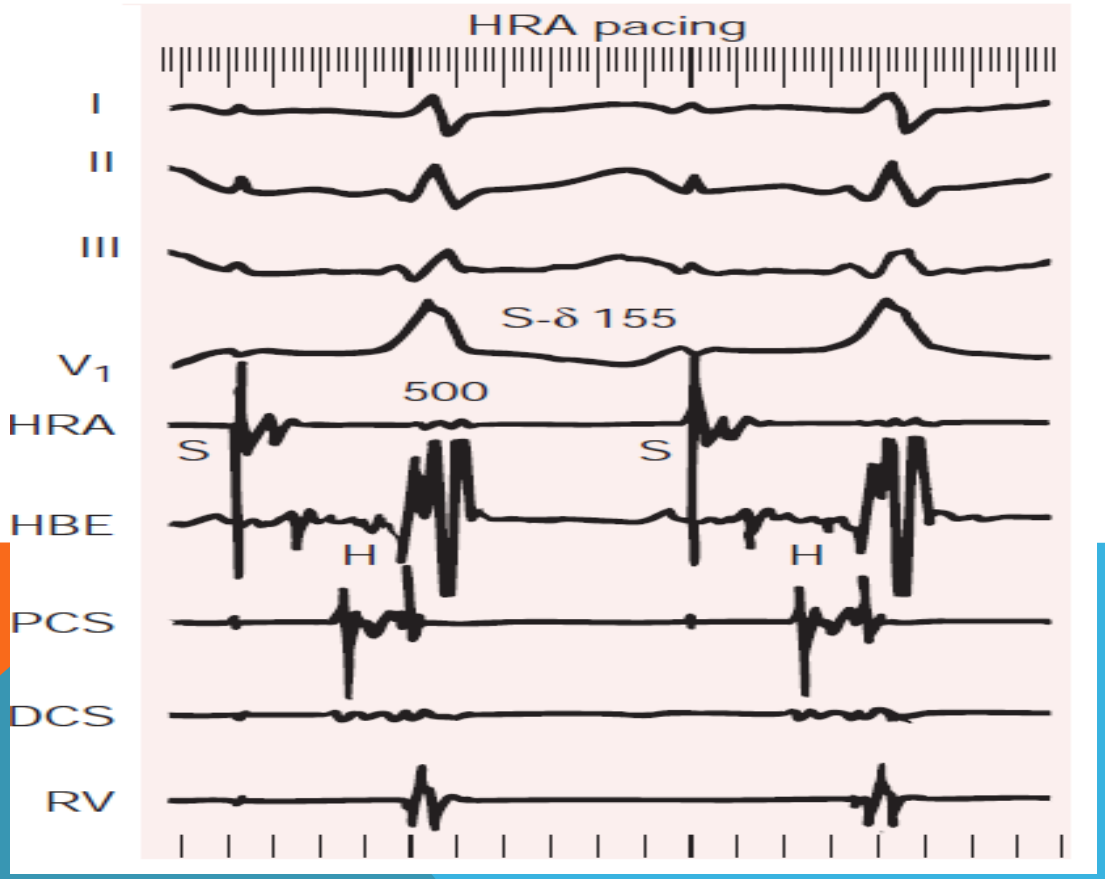
A right bundle branch block can be proarrhythmic by increasing the length of the tachycardia circuit (the VA interval is prolonged because of a delay in retrograde activation of the His bundle), and the tachycardia can become incessant.

In patients with fasciculoventricular connections, the H-V interval remains short and the QRS complex unchanged and anomalous during rapid atrial pacing.

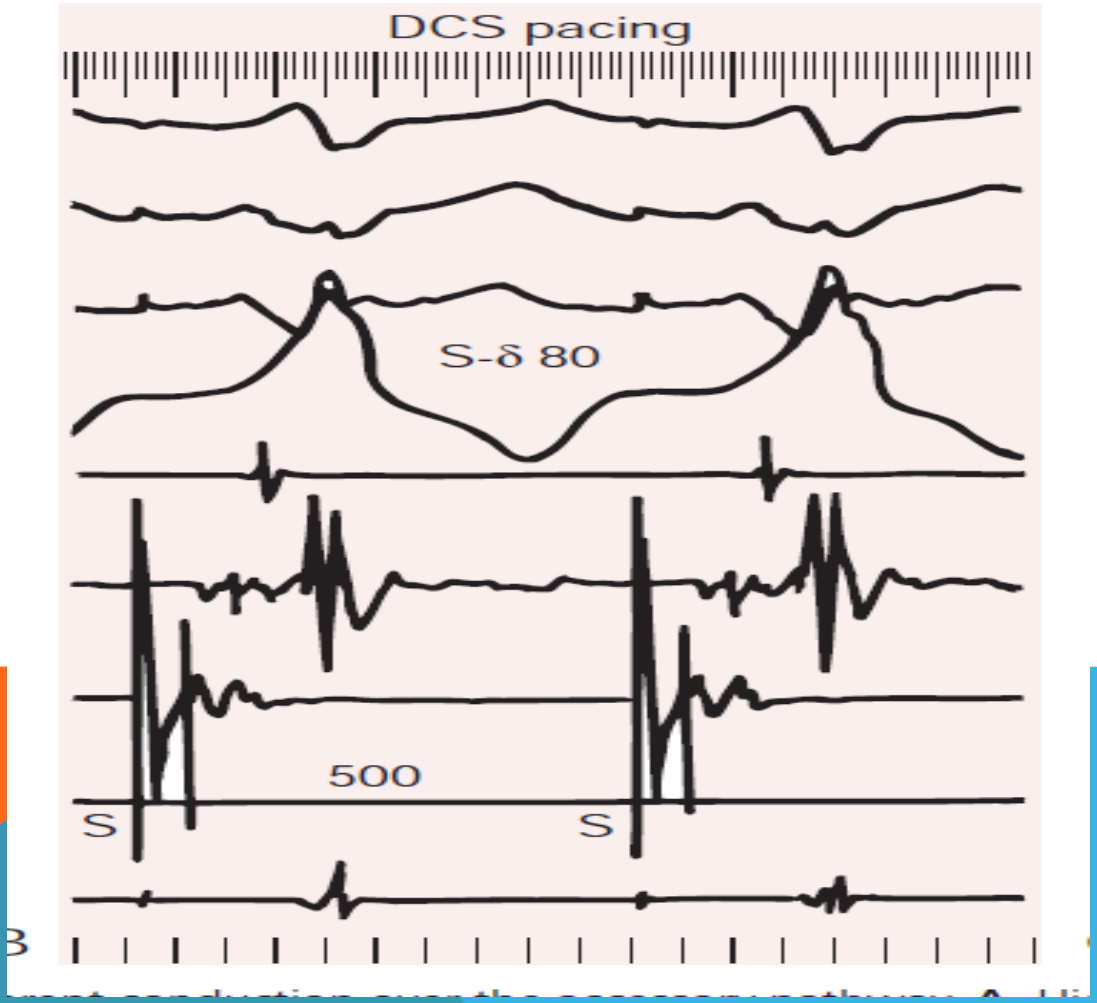
DEVELOPMENT OF PREEXCITATION OVER AN ATRIOFASCICULAR ACCESSORY PATHWAY. DURING ATRIAL PACING (S) ON THE LEFT SIDE OF THE FIGURE, CONDUCTION OCCURS DOWN THE AV NODE AS EVIDENCED BY A NORMAL-APPEARING QRS
COMPLEX AND A NORMAL H-V INTERVAL. THE STIMULUS MARKED BY THE *ARROWHEAD* CONDUCTS THE IMPULSE DOWN AN ATRIOFASCICULAR
FIBER, WHICH RESULTS IN A PREEXCITED QRS, AS EVIDENCED BY A WIDENED QRS AND SHORT H-V INTERVAL



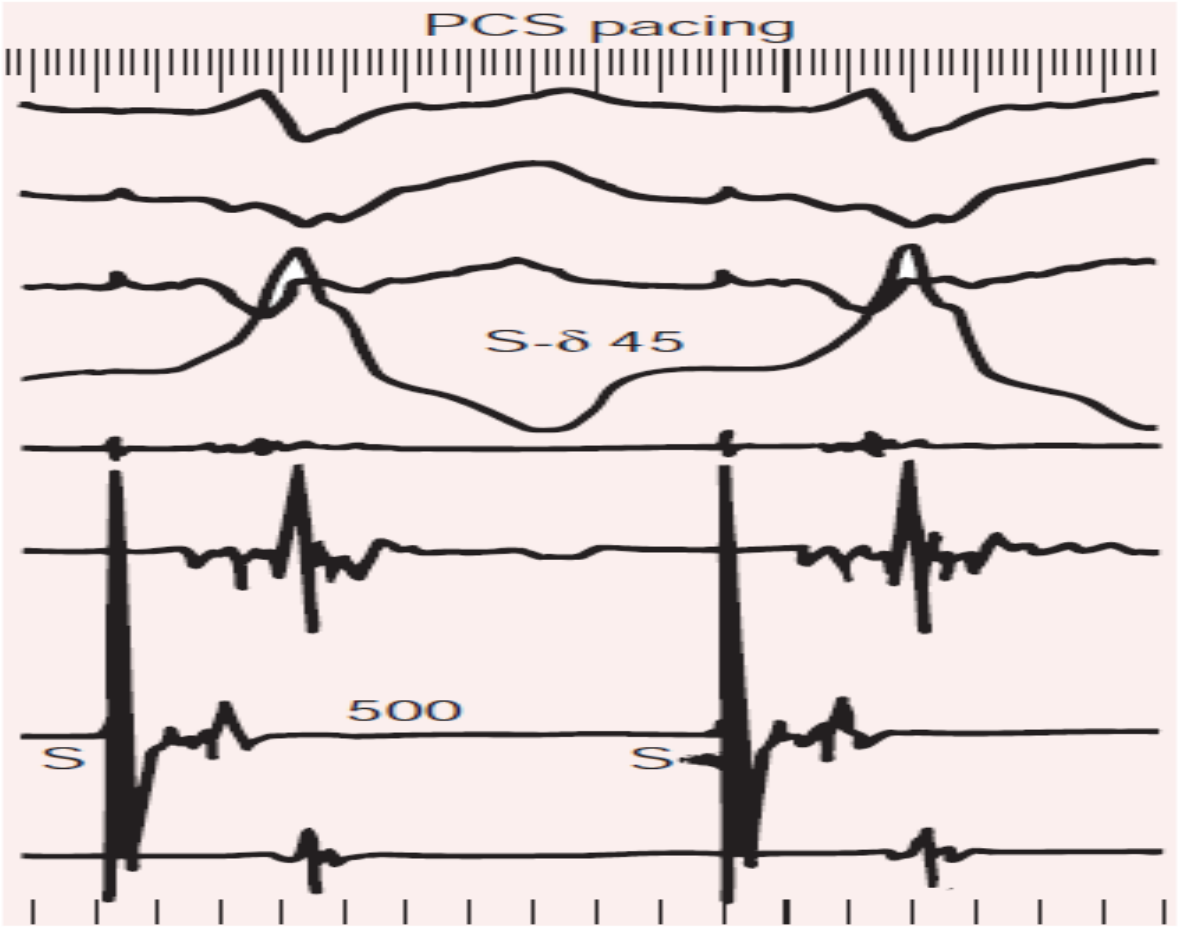
ATRIAL PACING AT DIFFERENT ATRIAL SITES ILLUSTRATING DIFFERENT CONDUCTION OVER THE ACCESSORY PATHWAY. **A**, HIGH RIGHTATRIAL (HRA) PACING AT A CYCLE LENGTH OF 500 MILLISECONDS PRODUCES ANOMALOUS ACTIVATION OF THE VENTRICLE (NOTE THE UPRIGHT QRS COMPLEX IN V1) AND A STIMULUS-DELTA INTERVAL OF 155 MILLISECONDS (S-Δ 155). THIS INTERVAL INDICATESTHAT THE TIME FROM THE ONSET OF THE STIMULUS TO THE BEGINNING OF THE QRS COMPLEX IS RELATIVELY LONG BECAUSE THE STIMULUS IS DELIVERED AT A FAIRLY LARGE DISTANCE FROM THE ACCESSORY PATHWAY. NOTE THAT HIS-BUNDLE ACTIVATION (H) OCCURS AT ABOUT THE ONSET OF THE QRS COMPLEX.



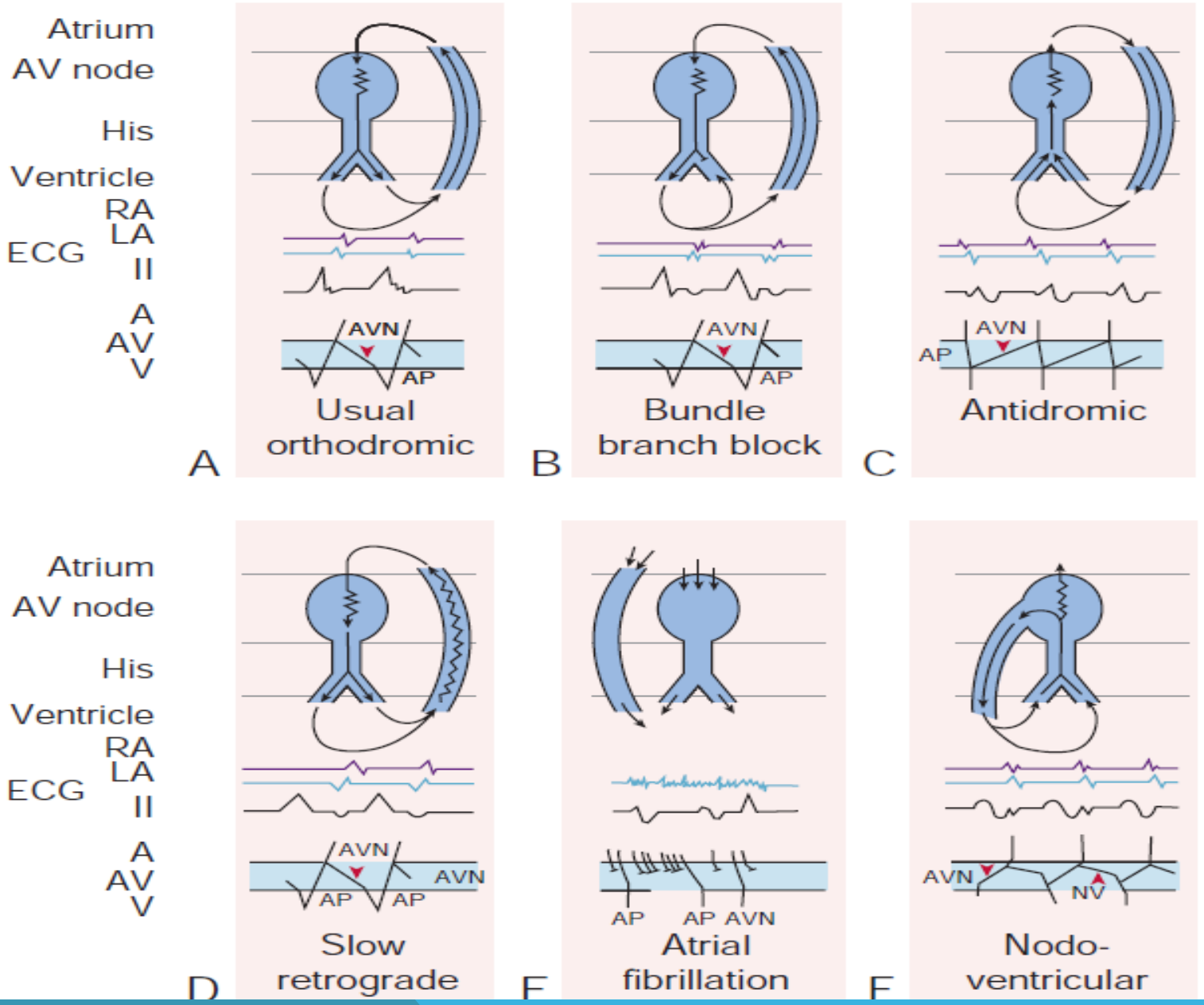
(DCS). AT THE SAME
PACING CYCLE LENGTH, DCS PACING RESULTS IN MORE ANOMALOUS VENTRICULAR ACTIVATION AND A SHORTER STIMULUS-DELTA INTERVAL (80 MSEC). HIS-BUNDLE ACTIVATION IS NOW BURIED WITHIN THE
INSCRIPTION OF THE VENTRICULAR ELECTROGRAM IN THE LOW RIGHT ATRIUM (HIS BUNDLE ELECTROGRAM [HBE] LEAD).



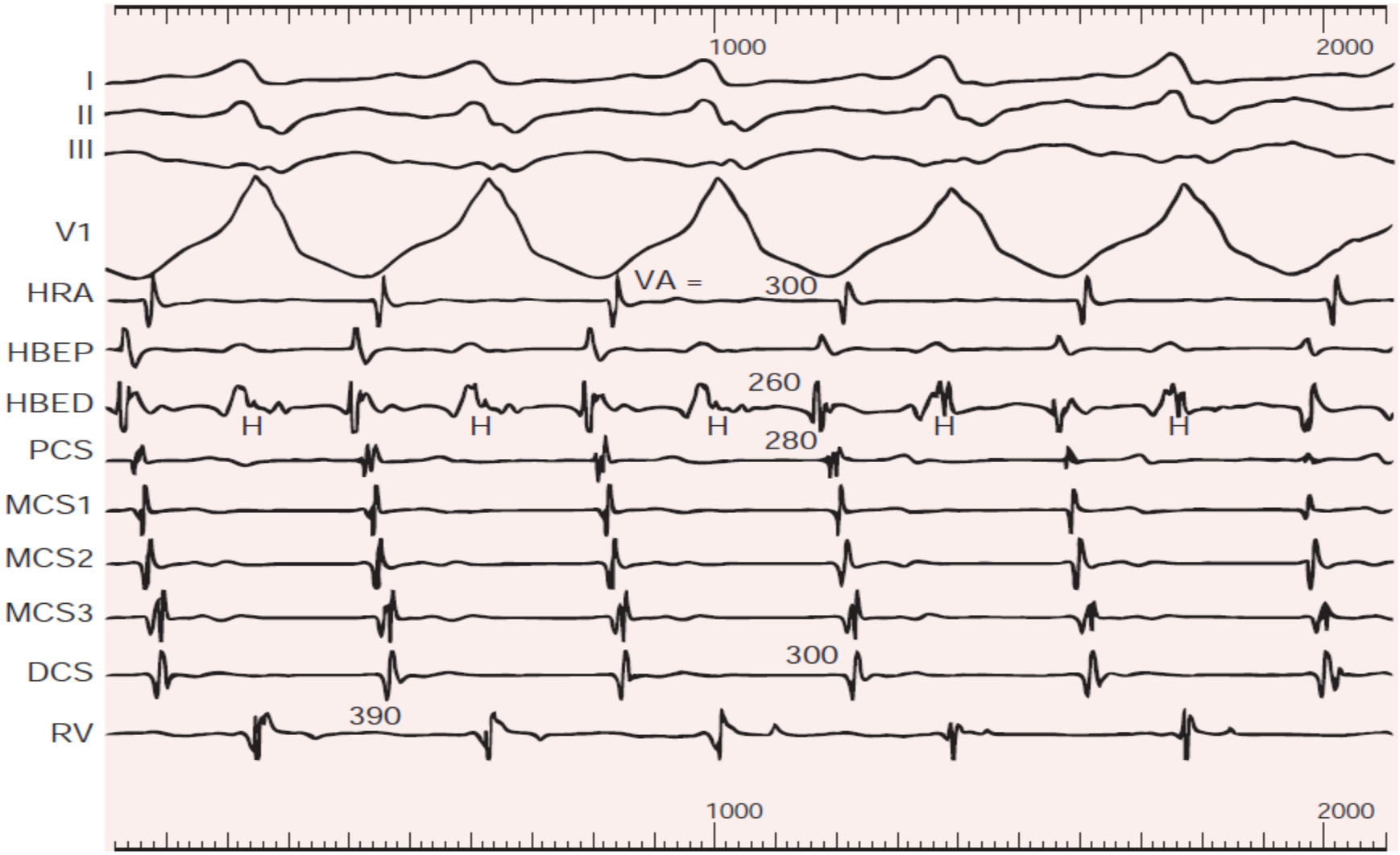
SHORTEST STIMULUS-DELTA INTERVAL (45 MSEC); SUCH AN INTERVAL INDICATES THAT THE PACING STIMULUS IS BEING DELIVERED VERY CLOSE TO THE ATRIAL INSERTION OF THE ACCESSORY PATHWAY, WHICH
IN THIS CASE IS LOCATED IN THE LEFT POSTEROSEPTAL REGION OF THE AV GROOVE



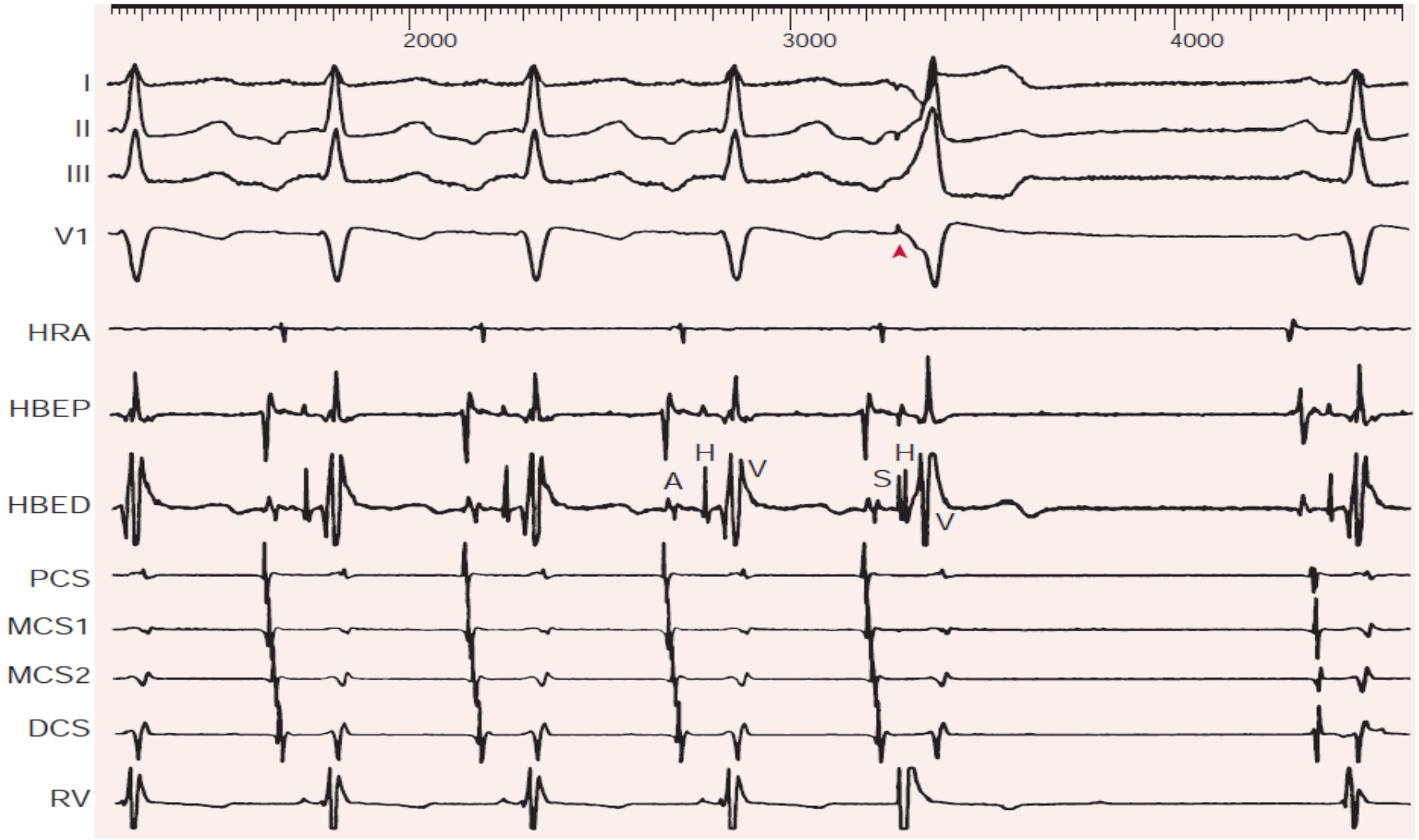
Reciprocating Tachycardias



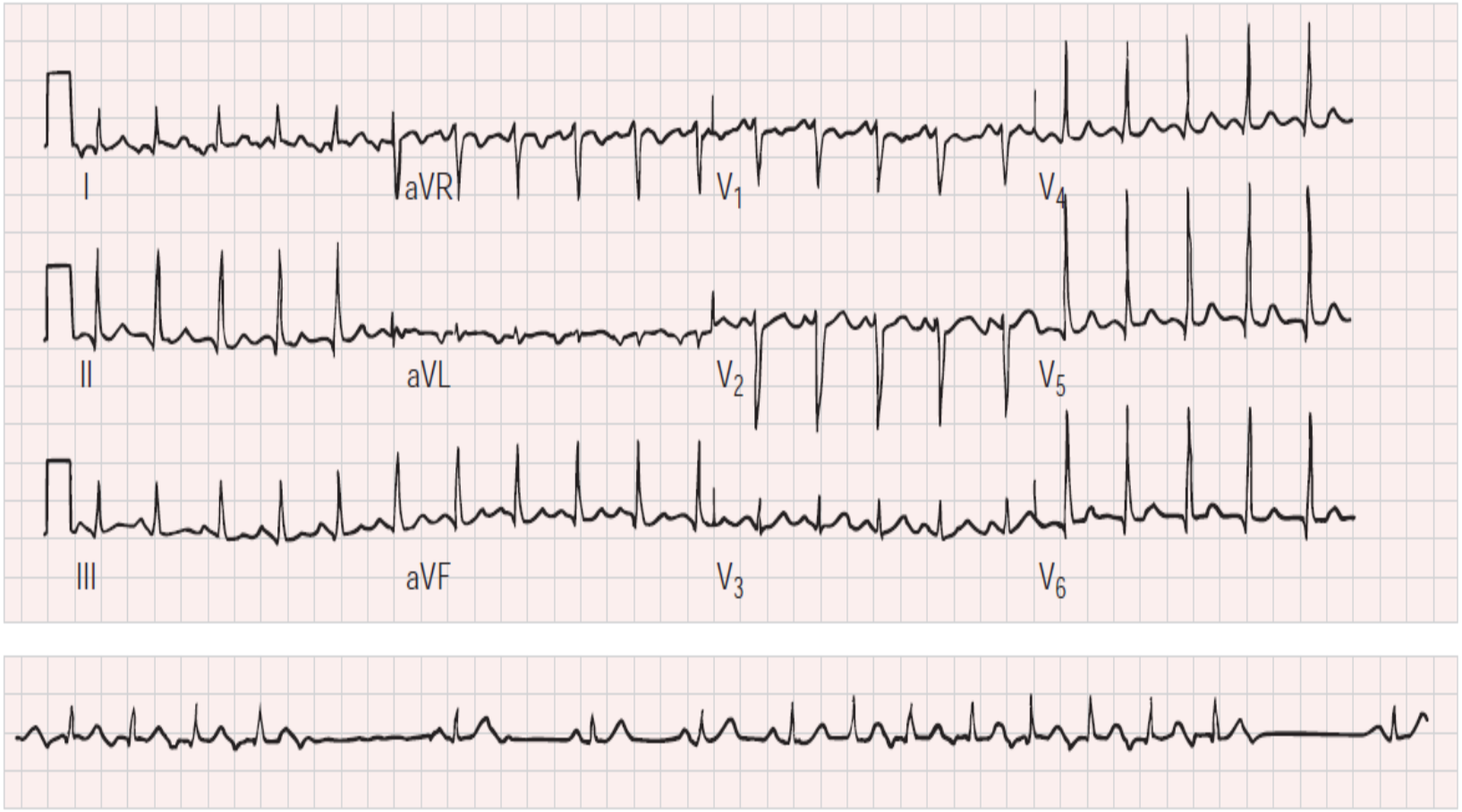
ANTIDROMIC AV RECIPROCATING TACHYCARDIA. THE TACHYCARDIA IN THIS EXAMPLE IS CAUSED BY ANTEROGRADE CONDUCTION OVER THE ACCESSORY PATHWAY (NOTE THE ABNORMAL QRS COMPLEX OF A LEFT POSTERIOR ACCESSORY PATHWAY) AND A NORMAL RETROGRADE ATRIAL ACTIVATION SEQUENCE (BEGINNING FIRST IN THE HBED LEAD), WHICH IS CAUSED BY RETROGRADE CONDUCTION OVER THE AV NODE. THE TACHYCARDIA CYCLE LENGTH IS 390 MILLISECONDS, WITH A VA INTERVAL OF 300 MILLISECONDS MEASURED IN THE HIGH RIGHT ATRIAL LEAD, 260 MILLISECONDS IN THE
DISTAL HIS LEAD, AND 280 MILLISECONDS IN THE PROXIMAL CORONARY SINUS LEAD. I, II, III, AND V1 ARE SCALAR LEAD



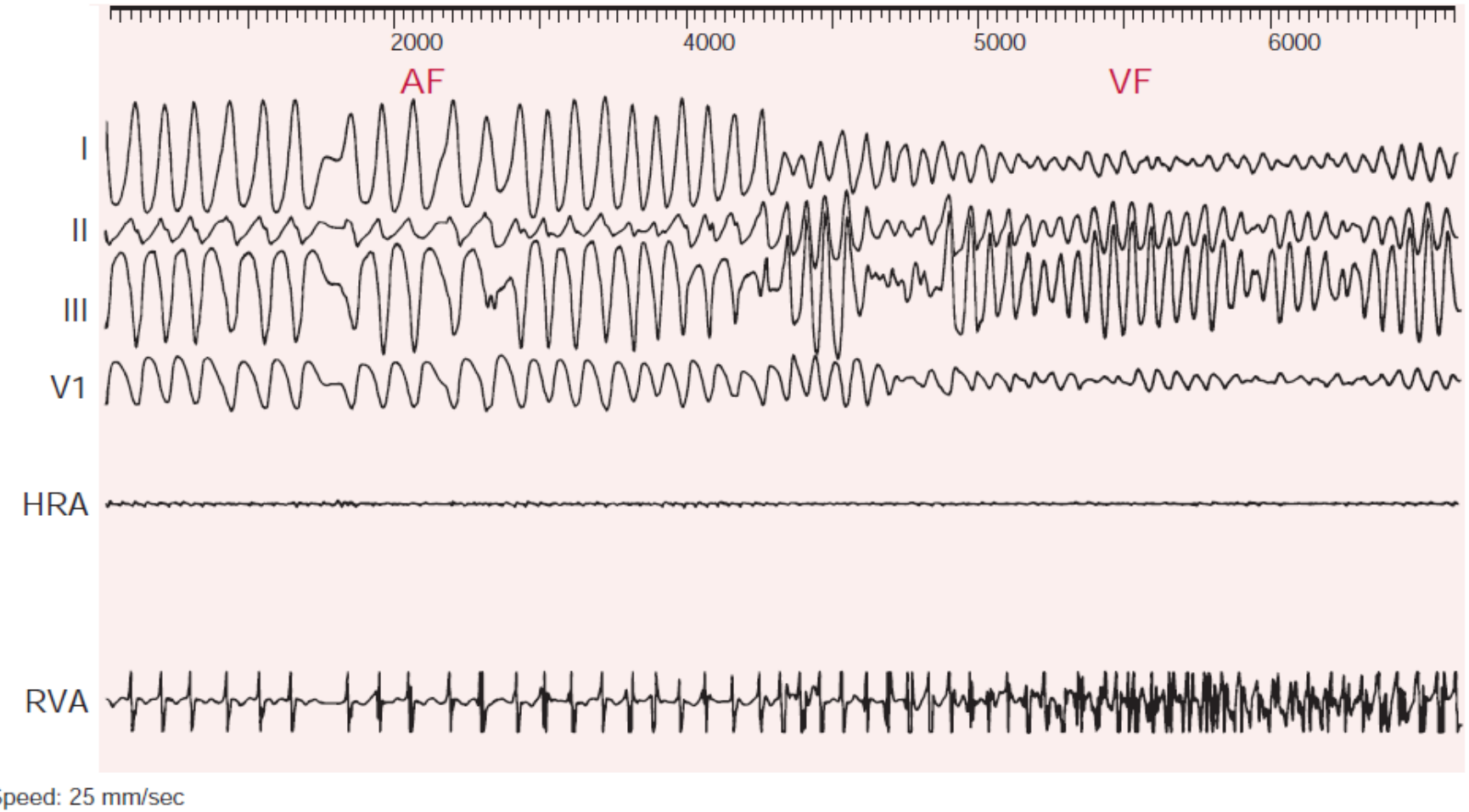
TERMINATION OF THE PERMANENT FORM OF AV JUNCTIONAL RECIPROCATING TACHYCARDIA (PJRT). IN THE LEFT PORTION OF THIS EXAMPLE, PJRT IS PRESENT. THE ATRIAL ACTIVATION SEQUENCE IS INDISTINGUISHABLE FROM ATYPICAL AV NODAL REENTRY AND ATRIAL TACHYCARDIA ORIGINATING IN THE LOW RIGHT ATRIUM. THE RESPONSE TO PREMATURE STIMULATION IDENTIFIES THE TACHYCARDIA AS PJRT. PREMATURE VENTRICULAR STIMULATION (*ARROWHEAD*) OCCURS AT A TIME WHEN THE HIS BUNDLE IS REFRACTORY FROM DEPOLARIZATION DURING THE TACHYCARDIA (SECOND LABELED H). THEREFORE PREMATURE VENTRICULAR STIMULATION CANNOT ENTER THE AV NODE. FURTHERMORE, PREMATURE VENTRICULAR STIMULATION DOES NOT REACH THE ATRIUM. PREMATURE VENTRICULAR STIMULATION, HOWEVER, TERMINATES THE TACHYCARDIA. THIS DETAIL CAN BE EXPLAINED ONLY BY THE PVC INVADING AND BLOCKING IN A RETROGRADELY CONDUCTING ACCESSORY PA



PATIENT WITH A LEFT-SIDED ACCESSORY PATHWAY. THE 12-LEAD ECG DEMONSTRATES A LONG RP INTERVAL–SHORT PR INTERVAL TACHYCARDIA, WHICH IN CONTRAST TO THE USUAL FORM OF PJRT, EXHIBITS NEGATIVE P WAVES IN LEADS I AND AVL. THE RHYTHM STRIPS BELOW (LEAD I) INDICATE THAT WHENEVER A NONCONDUCTED P WAVE OCCURS, THE TACHYCARDIA ALWAYS TERMINATES, ONLY TO BEGIN AGAIN AFTER SEVERAL SINUS BEATS.



THIS PANEL, THE ECG DEMONSTRATES AF WITH CONDUCTION OVER AN ACCESSORY PATHWAY PRODUCING A RAPID VENTRICULAR RESPONSE, AT TIMES IN EXCESS OF 390 BEATS/MIN. IN THE MIDPORTION OF THE TRACING, VF CAN BE SEEN TO DEVELOP.



TREATMENT

Young patients (8 to 21 years of age) who have only persistent electrocardiographic abnormalities, without tachyarrhythmias or a history of palpitations, should undergo stress testing to determine whether abrupt loss of preexcitation occurs. If loss of preexcitation does not occur or is equivocal or not abrupt, an invasive EP study is recommended to further risk-stratify patients.

FOR PATIENTS WITH FREQUENT EPISODES OF SYMPTOMATIC TACHYARRHYTHMIA, THERAPY SHOULD BE INITIATED.

TABLE 37-3 Drugs That Slow Conduction in and Prolong Refractoriness of the Accessory Pathway and Atrioventricular Node

AFFECTED TISSUE	DRUGS
Accessory pathway	Class IA
AV node	Class II Class IV Adenosine Digitalis
Both	Class IC Class III (amiodarone)

For long-term therapy to prevent recurrence, RF catheter ablation of the accessory pathway has become the first-line therapy for most patients.

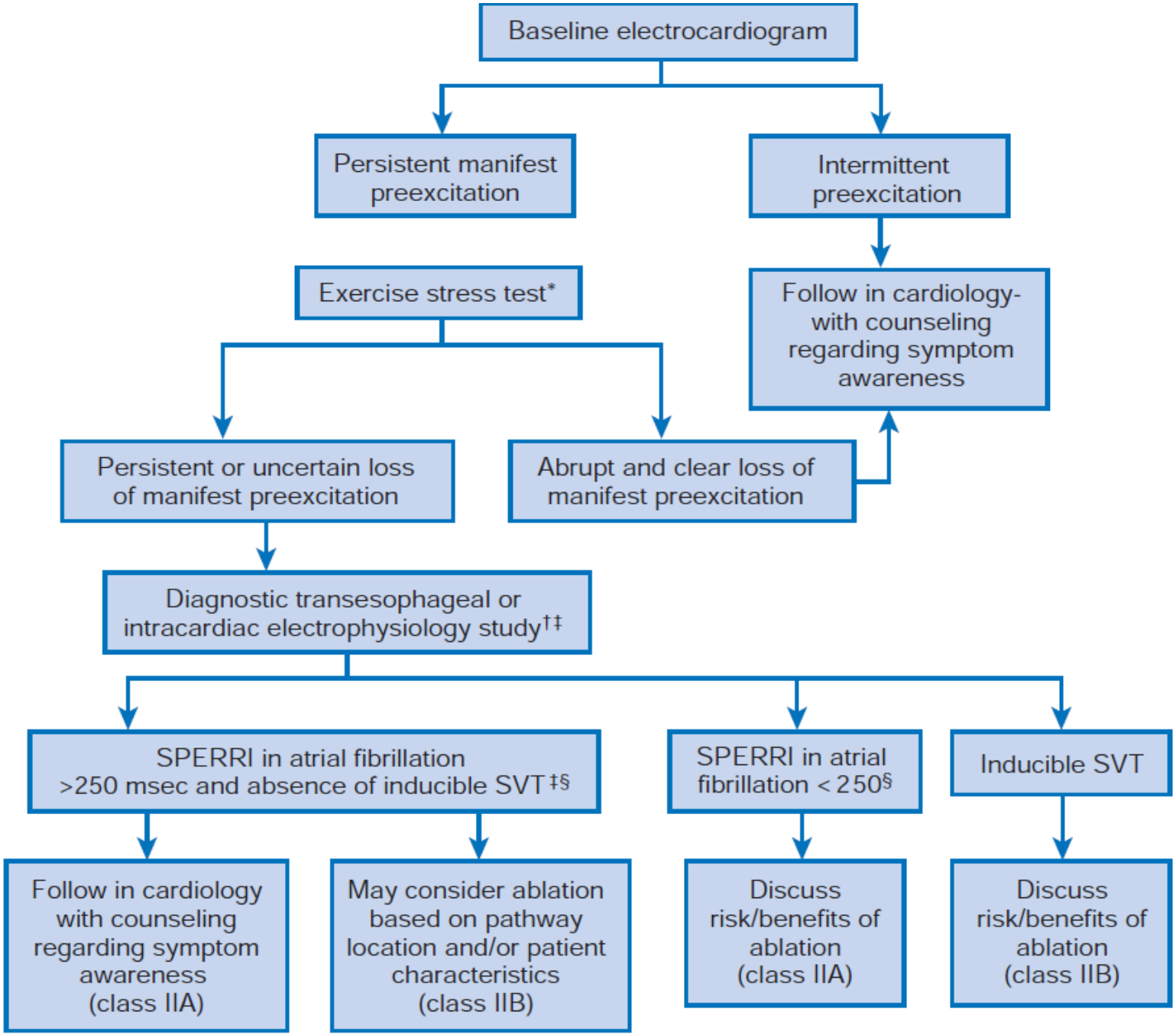

Baseline electrocardiogram
Persistent manifest preexcitation
Intermittent preexcitation
Exercise stress test*
Follow in cardiology-with counseling regarding symptom awareness
Persistent or uncertain loss of manifest preexcitation
Abrupt and clear loss of manifest preexcitation
Diagnostic transesophageal or intracardiac electrophysiology study†‡
SPERRI in atrial fibrillation >250 msec and absence of inducible SVT‡§
SPERRI in atrial fibrillation < 250§
Inducible SVT
Follow in cardiology with counseling regarding symptom awareness (class IIA)
May consider ablation based on pathway location and/or patient characteristics (class IIB)
Discuss risk/benefits of ablation (class IIA)
Discuss risk/benefits of ablation (class IIB)

TABLE 37-5 Supraventricular Tachycardias

SHORT RP, LONG PR INTERVAL	LONG RP, SHORT PR INTERVAL
AV nodal reentry	Atrial tachycardia
AV reentry	Sinus node reentry Atypical AV nodal reentry AVRT with a slowly conducting accessory pathway (e.g., PJRT)

ATRIOFASCICULAR (MAHAIM FIBER) AP

The proximal insertion of the pathway is at the atrial margin of the free wall TV annulus and the distal insertion either at the RBB (atriofascicular) or directly into ventricular myocardium(AV).

During sinus rhythm they demonstrate only minimal preexcitation.

These Aps conduct only in the antegrade direction,& exhibit long baseline conduction time, decremental conduction,Wenckebach behavior,and trnsient conduction block with adenosin.

With very rare exceptions ,these pathways are right-sided.

They directly participate in antidromic reciprocating tachycardia only.

Atrium>> AP >>RBB>>His >>AVN >>atrium

DIAGNOSIS

These pt.. Can be young with or without cardiac structural abnormality.

Among all AP atriofascicular pathways are relatively uncommon,(2-3%)

ECG:

Little or no sign.

Minimal ventricular preexcitation may be suggested by the absence of septal Q waves in leads:I,aVL ,V5&V6. or the presence of an rS QRS in lead III

During preexcitation ,the QRS axis is between 0 and -75 degrees.QRS width is 0.15 seconds or less , QRS transition occurs after V4 .

QRS morphology : LBBB

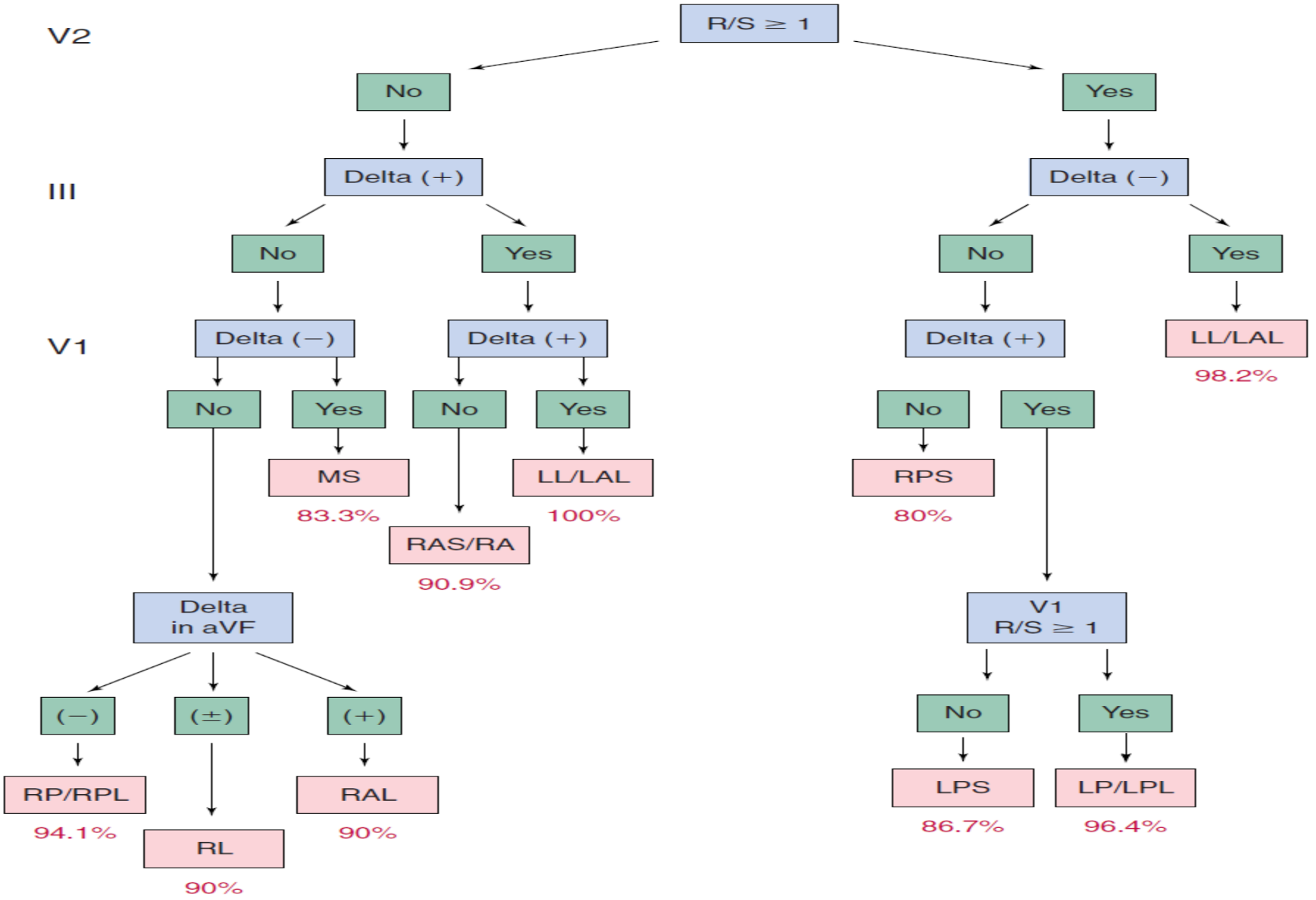
FASCICULOVENTRICULAR AP

HV is less than 35ms

The hallmark finding is a fixed degree of preexcitation with all atrial cycle lengths

Continued presence of preexcitation during pure His pacing is pathognomonic

Don’t require ablation


V2
III
V1
R/S ≥ 1
No
Yes
Delta (+)
Delta (−)
No
Yes
No
Yes
Delta (−)
Delta (+)
Delta (+)
LL/LAL
98.2%
No
Yes
No
Yes
No
Yes
MS
83.3%
RAS/RA
90.9%
LL/LAL
100%
RPS
80%
Delta in aVF
V1 R/S ≥ 1
(−)
(±)
(+)
No
Yes
RP/RPL
94.1%
RL
90%
RAL
90%
LPS
86.7%
LP/LPL
96.4%

VENTRICULAR RHYTHM DISTURBANCES

Premature Ventricular Complexes:

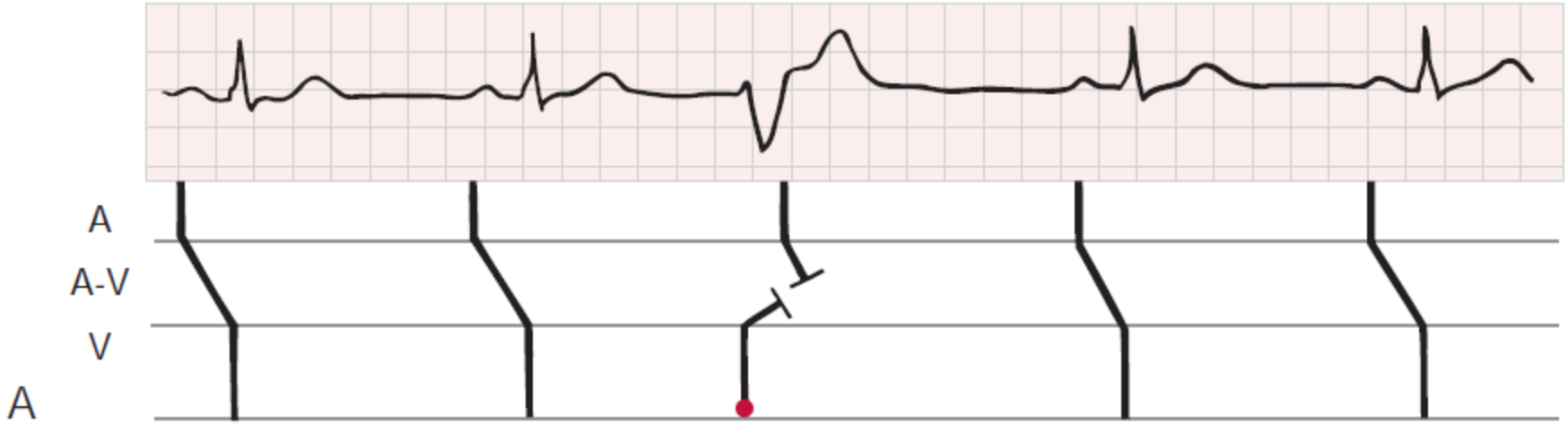
premature occurrence of a QRS

Exceeding the dominant QRS complex

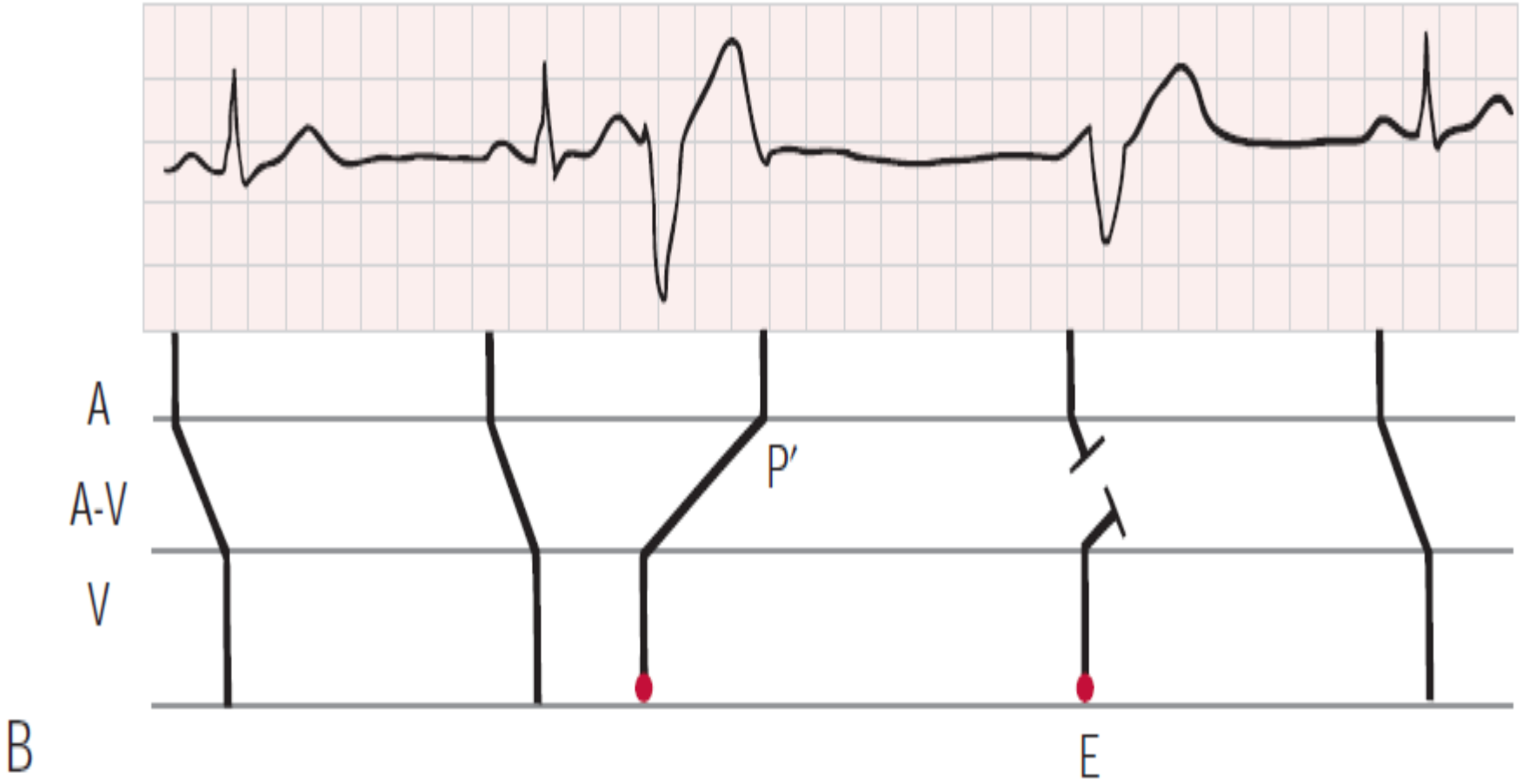
T wave is usually large and opposite

QRS complex is not preceded by a premature P wave

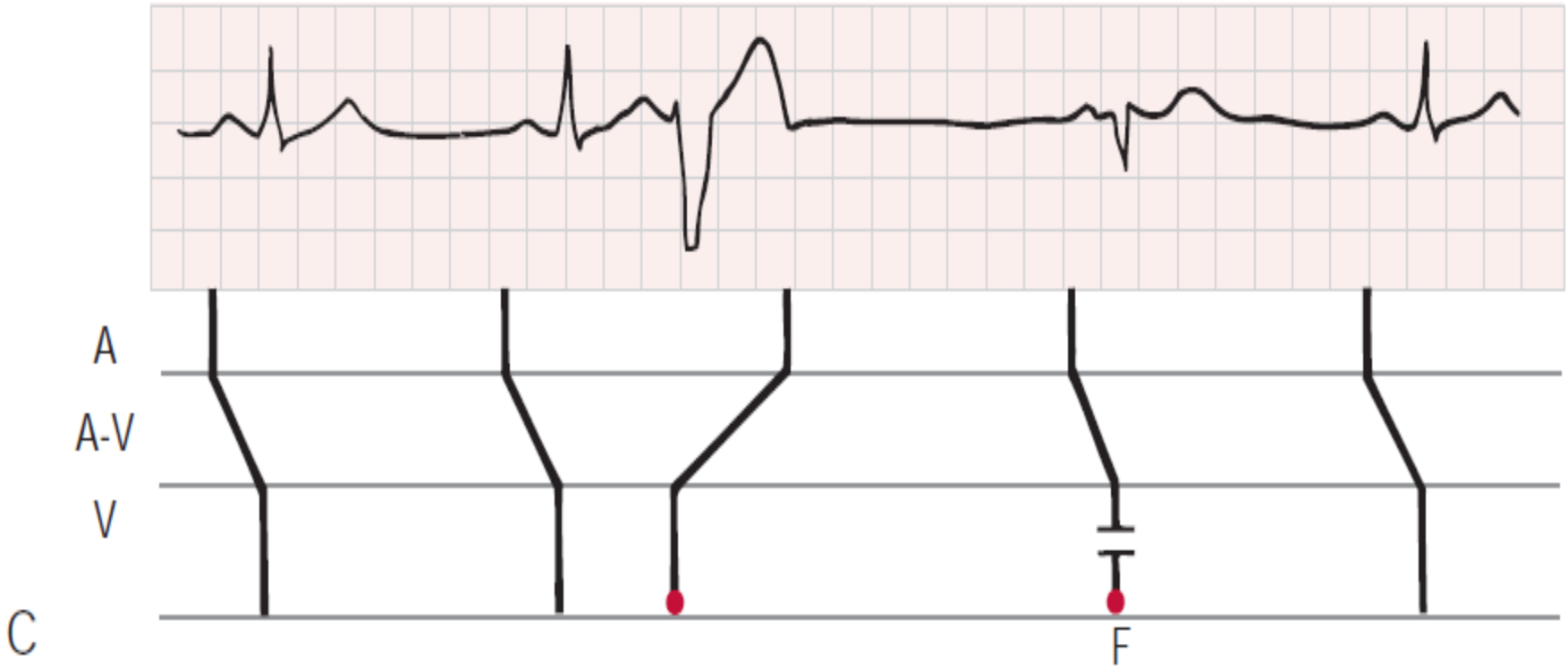
PVC COMPENSATORY PAUSE



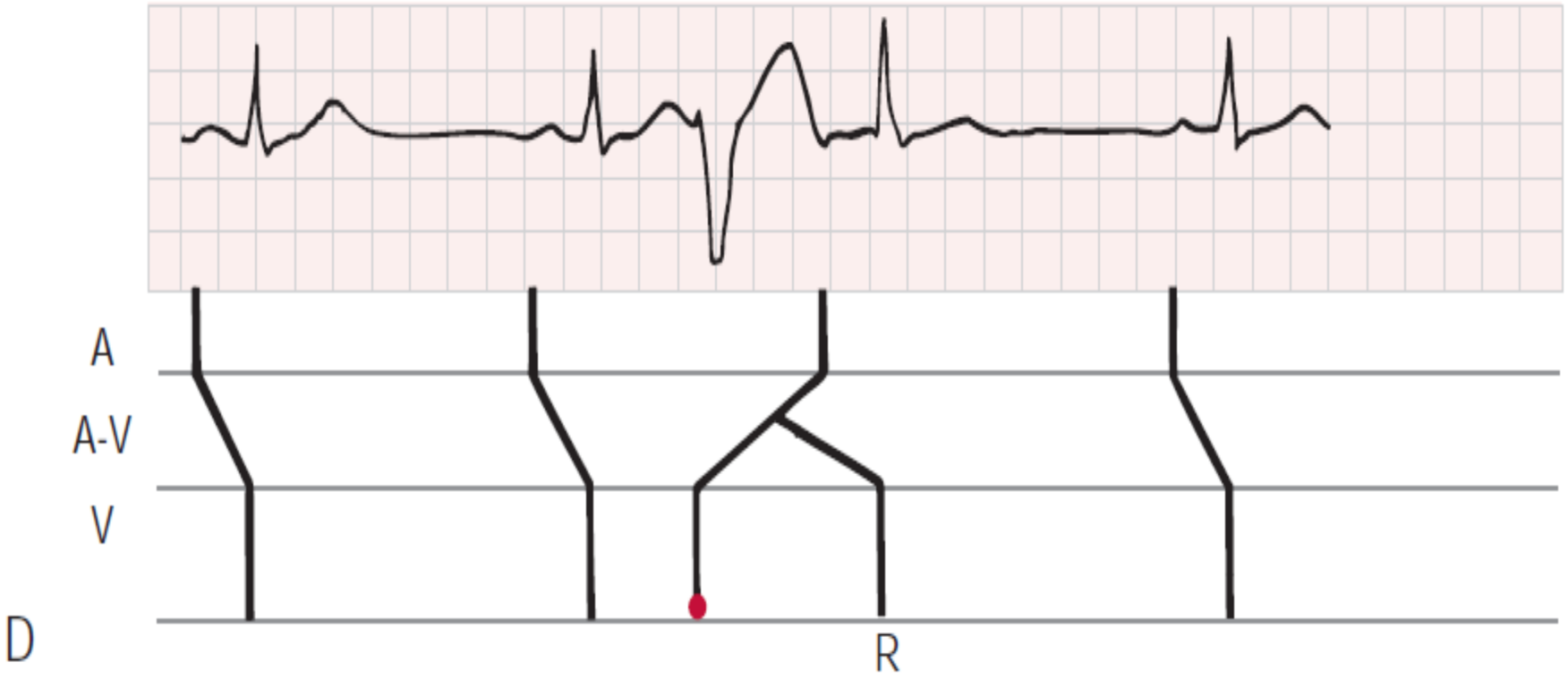
PVC NONCOMPENSATORY PAUSE



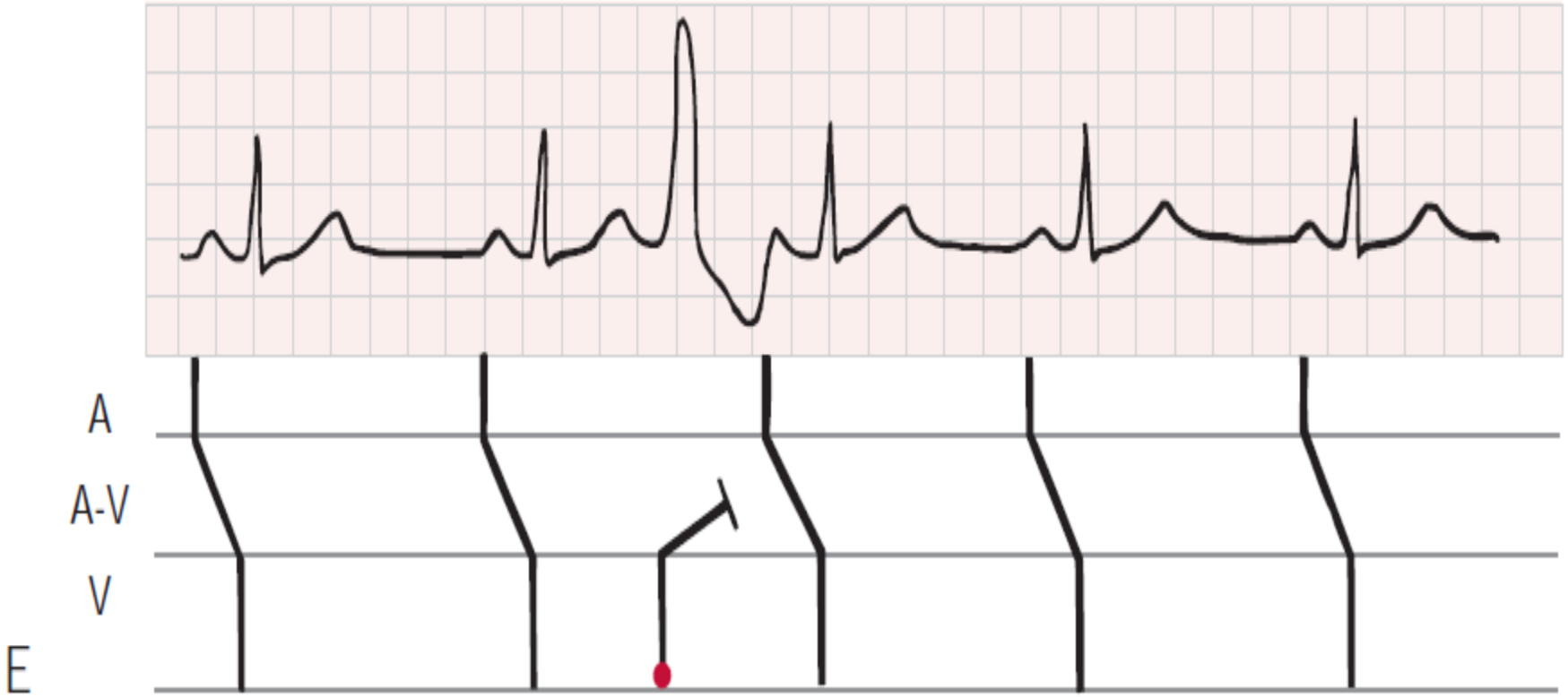
PVC VENTRICULAR FUSION BEAT



PVC VENTRICULAR ECHO



PVC INTERPOLATED



MULTIFORM PVCS

V_1

CLINICAL FEATURES

The prevalence of premature complexes increases with age, male sex,andhypokalemia.

Long runs of frequent PVCs in patients with heart disease can produce angina, hypotension, or heart failure.

Premature systoles can be very uncomfortable in patients with aortic regurgitation because of their large stroke volume.

PVCs occur in association with various stimuli and can be produced by direct mechanical, electrical, and chemical stimulation of the myocardium. Frequently, they are noted in patients with left ventricular (LV) false tendons, during infection, in ischemic or inflamed myocardium, and during hypoxia, anesthesia, or surgery

PHYSICAL EXAMINATION

decrease in intensity of the heart sounds

decreased or absent peripheral (e.g., radial) pulse.

The relationship of atrial to ventricular systole determines the presence of normal *a* waves or giant *a* waves in the jugular venous pulse

the length of the PR interval determines the intensity of the first heart sound.

The second heart sound can be split abnormally,(S2 sppliting) depending on the origin of the ventricular complex.

THE IMPORTANCE OF PVCS DEPENDS ON THE CLINICAL SETTING

Frequent PVCs can lead to LV dysfunction over time.

Features predicting the development of PVC-induced cardiomyopathy:

1. PVC burden greater than 24%
2. very wide-QRS PVCs
3. PVCs of epicardial origin.

Ablation generally resolves the cardiomyopathy,

MANAGEMENT

In most patients, PVCs do not need to be treated

PVCs accompanying slow ventricular rates can be abolished by increasing the basic rate with atropine or isoproterenol or by pacing,

slowing of the heart rate in some patients with sinus tachycardia can eradicate PVCs.

Frequent PVCs, even in the setting of acute myocardial infarction, need not be treated unless they directly contribute to hemodynamic compromise, which is very rare.

Beta blockers are often the first line of therapy

For patients with significant symptoms, particularly those with reduced cardiac function, RF ablation of the PVC focus can be effective and improve cardiac performance.

Low levels of serum potassium and magnesium are associated with higher prevalence rates of ventricular arrhythmias.

ACCELERATED IDIOVENTRICULAR RHYTHM

The ventricular rate, commonly between 60 and 110 beats/minute,

fusion beats often occur at the onset and termination of the arrhythmia as the pacemakers vie for control of ventricular depolarization

Because of the slow rate, capture beats are common

The onset of this arrhythmia is generally gradual (nonparoxysmal) and occurs when the rate of the VT exceeds the sinus rate as a result of sinus slowing or SA or AV block.

Termination of the rhythm generally occurs gradually as the dominant sinus rhythm accelerates or as the ectopic ventricular rhythm decelerates

MANY CHARACTERISTICS INCRIMINATE
ENHANCED AUTOMATICITY AS THE RESPONSIBLE MECHANISM.

The arrhythmia occurs as a rule in patients who have heart disease,

such as those with acute myocardial infarction or digitalis toxicity

Does not appear to seriously affect the patient's clinical

course or the prognosis.

It commonly occurs at the moment of reperfusion of a previously occluded coronary artery and can be found during resuscitation.

MANAGEMENT

Suppressive therapy is rarely necessary because the ventricular rate is generally less than 100 beats/minute

simply increasing the sinus rate with atropine or atrial pacing suppresses the accelerated idioventricular rhythm

VENTRICULAR TACHYCARDIA

VT arises distal to the bifurcation of the His bundle in the specialized

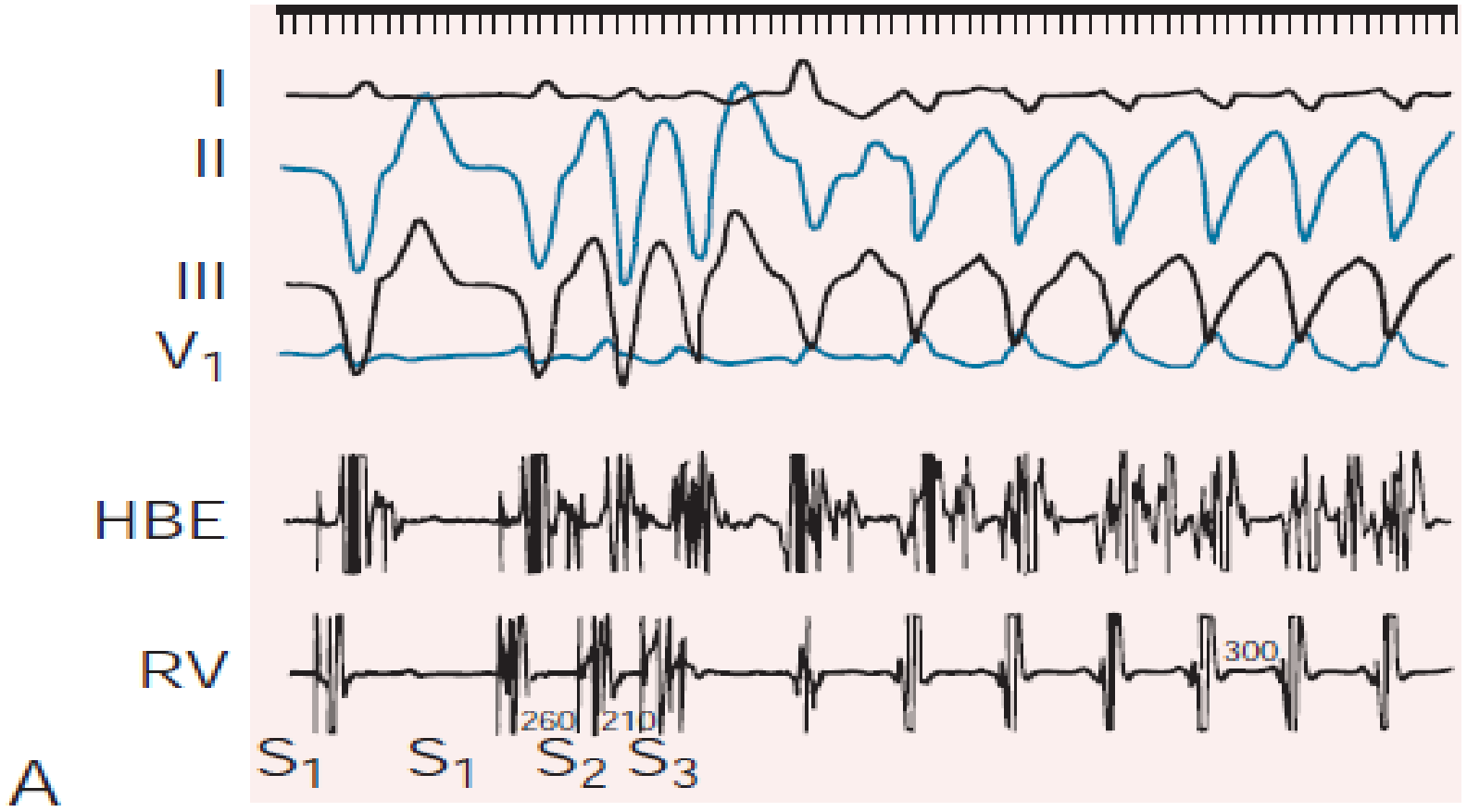
conduction system, ventricular muscle, or combinations of both types of tissue.

Mechanisms include disorders of impulse formation (enhanced automaticity or triggered activity) and conduction (reentry)

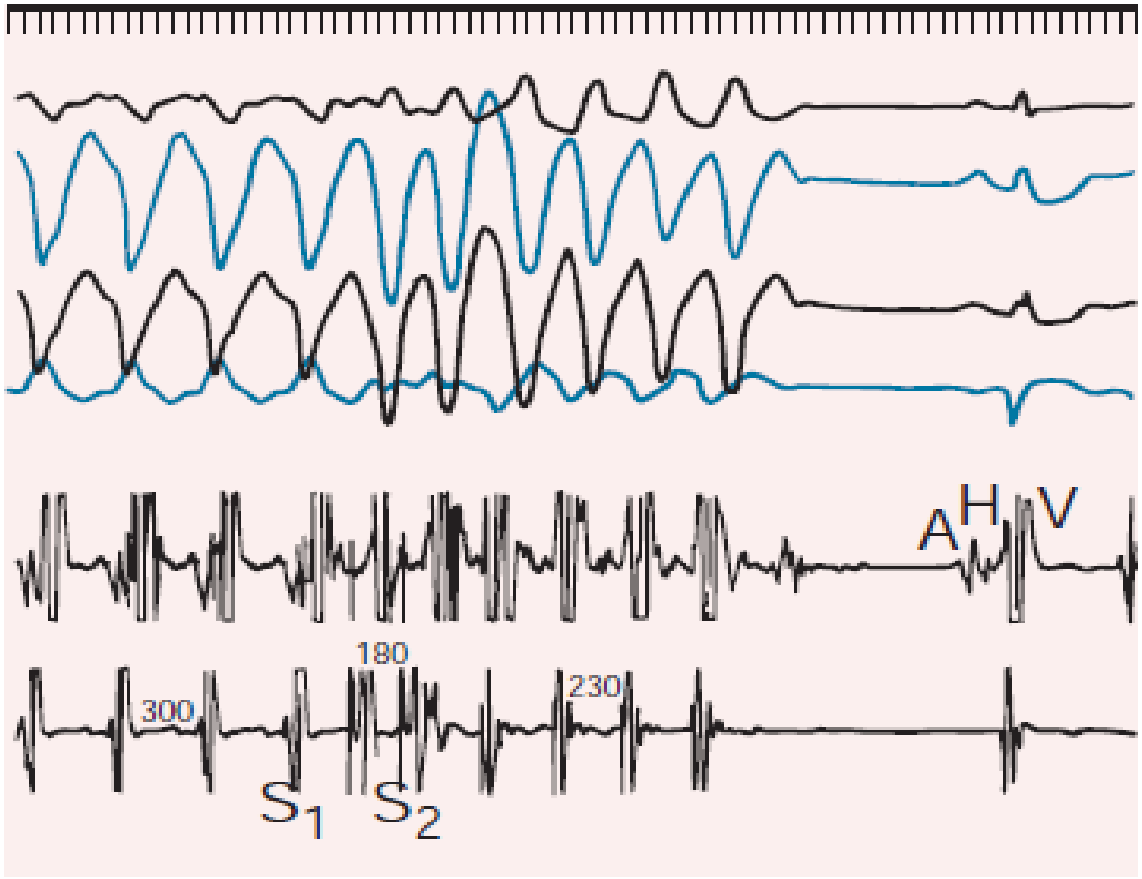
In general, the specific type, prognosis, and management of VT depend on whether

underlying structural heart disease is present.

A PREMATURE STIMULUS (S2) AT AN S1-S2 INTERVAL OF 260 MILLISECONDS AND ANOTHER PREMATURE STIMULUS (S3) AT A CYCLE LENGTH OF 210 MILLISECONDS INITIATE SUSTAINED MONOMORPHIC VT AT A CYCLE LENGTH OF 300 MILLISECONDS.



TWO PREMATURE
VENTRICULAR STIMULI (S1-S2) CREATE AN UNSTABLE VT THAT PERSISTS FOR SEVERAL BEATS
AT A SHORTER CYCLE LENGTH (230 MSEC) AND THEN TERMINATES, FOLLOWED BY SINUS RHYTHM



B

Patients can have VTs with multiple morphologies originating at the same or closely adjacent sites, probably with different exit paths.

Others have multiple sites of origin.

Atrial activity can be independent of ventricular activity, or the atria can be depolarized by the ventricles retrogradely (VA association)

Depending on the particular type of VT, rates range from 70 to 250 beats/minute, and the onset can be paroxysmal (sudden) or nonparoxysmal

QRS CONTOURS DURING THE VT CAN BE:

unchanging (uniform, monomorphic)

can vary randomly (multiform, polymorphic, or pleomorphic):

in a more or less repetitive manner (torsades de pointes),

in alternate complexes (bidirectional VT)

in a stable but changing contour (i.e., right bundle branch contour changing to a left bundle branch contour)

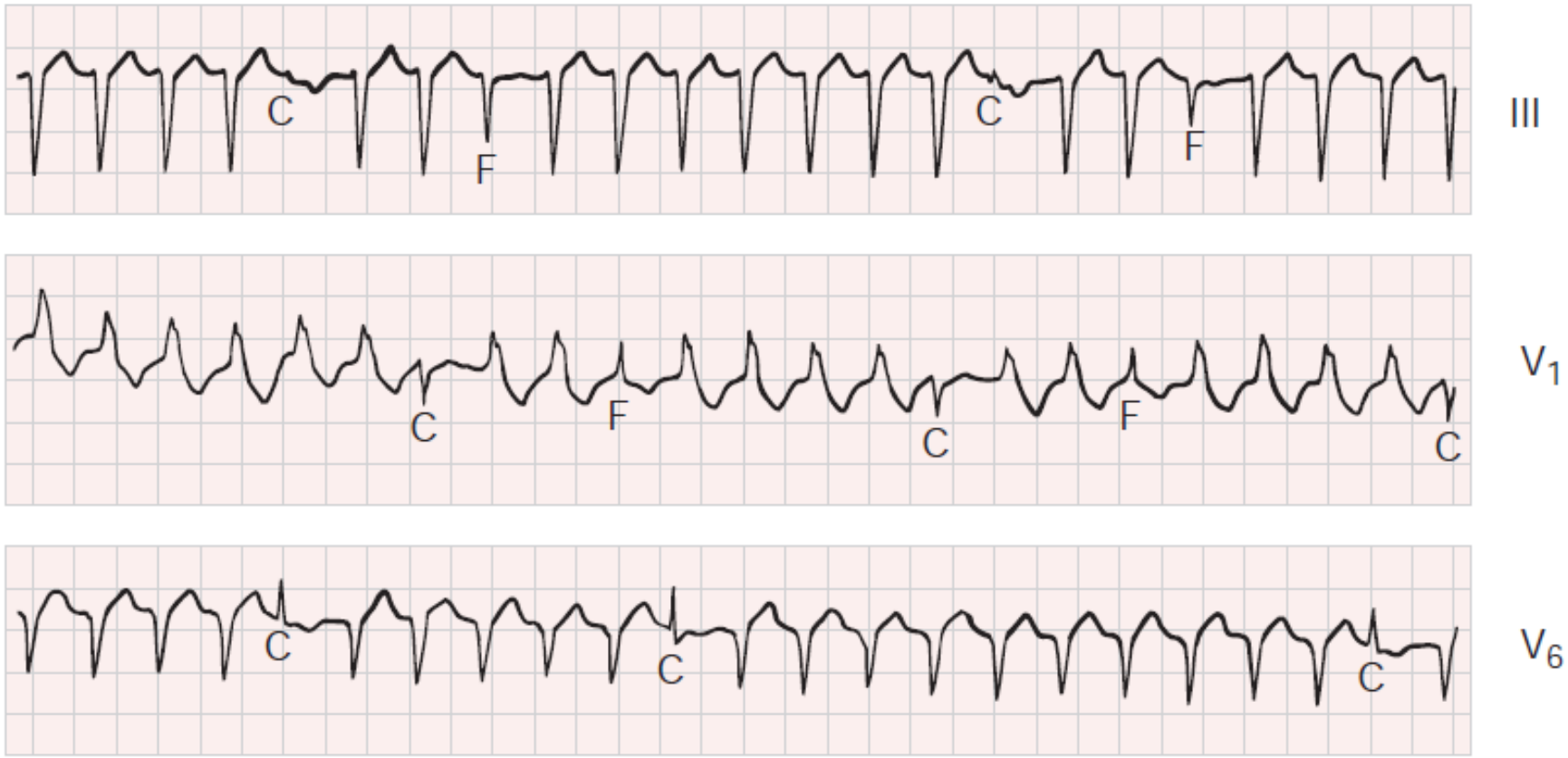
VT can be sustained, defined arbitrarily as lasting longer than 30 seconds or requiring termination because of hemodynamic collapse,

Can be nonsustained, when it stops spontaneously in less than 30 seconds.

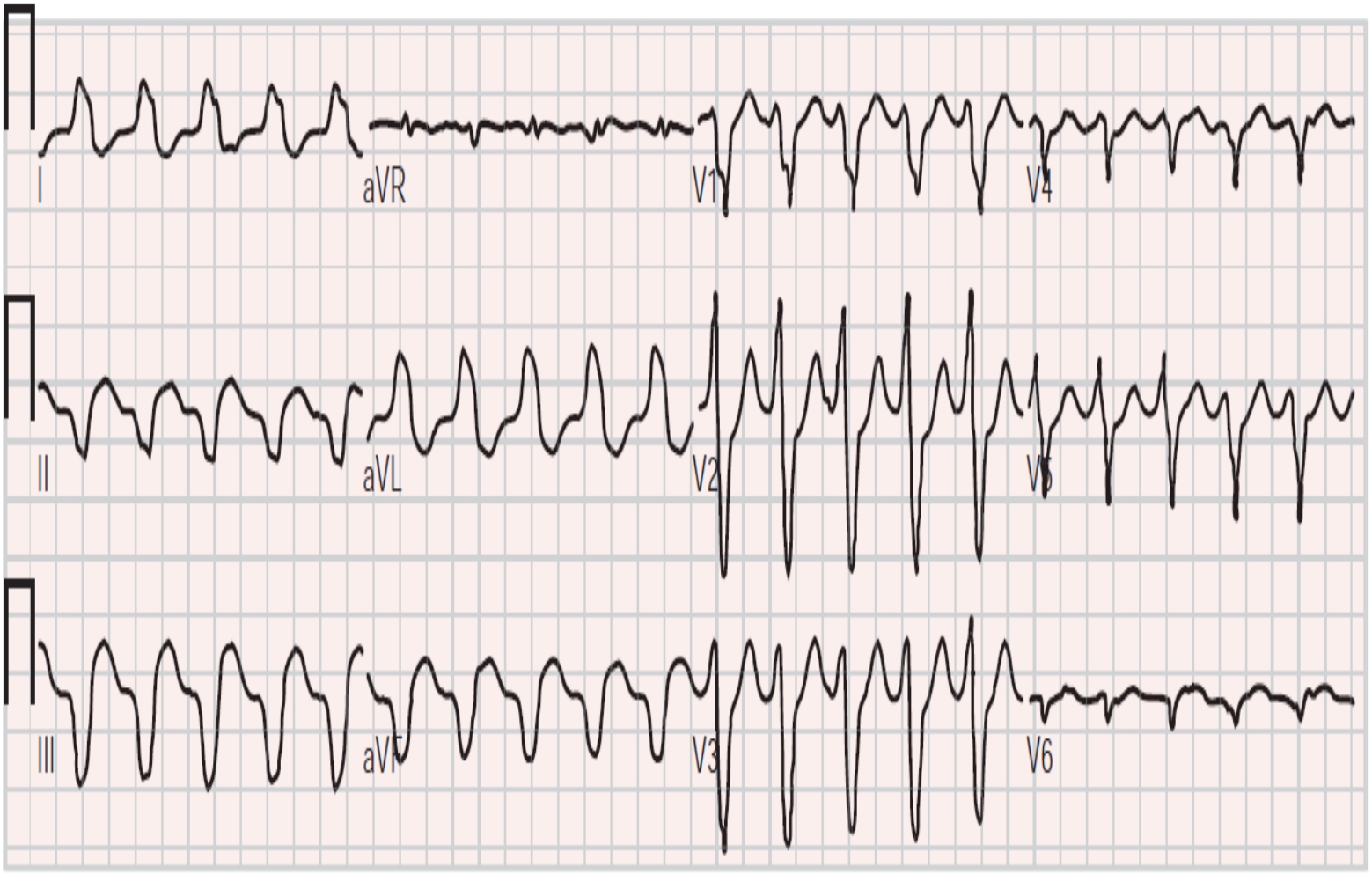
MAJOR FEATURES IN THE DIFFERENTIAL DIAGNOSIS OF WIDE-QRS BEATS VERSUS TACHYCARDIA

SUPPORTS SVT	SUPPORTS VT
Slowing or termination by vagal tone	Fusion beats
Onset with premature P wave	Capture beats
RP interval ≤100 msec	AV dissociation
P and QRS rate and rhythm linked to suggest that ventricular activation depends on atrial discharge, e.g., 2:1 AV block rSR′ V_1	P and QRS rate and rhythm linked to suggest that atrial activation depends on ventricular discharge, e.g., 2:1 VA block
Long-short cycle sequence	"Compensatory" pause
	Left-axis deviation; QRS duration >140 msec
	Specific QRS contours (see text)

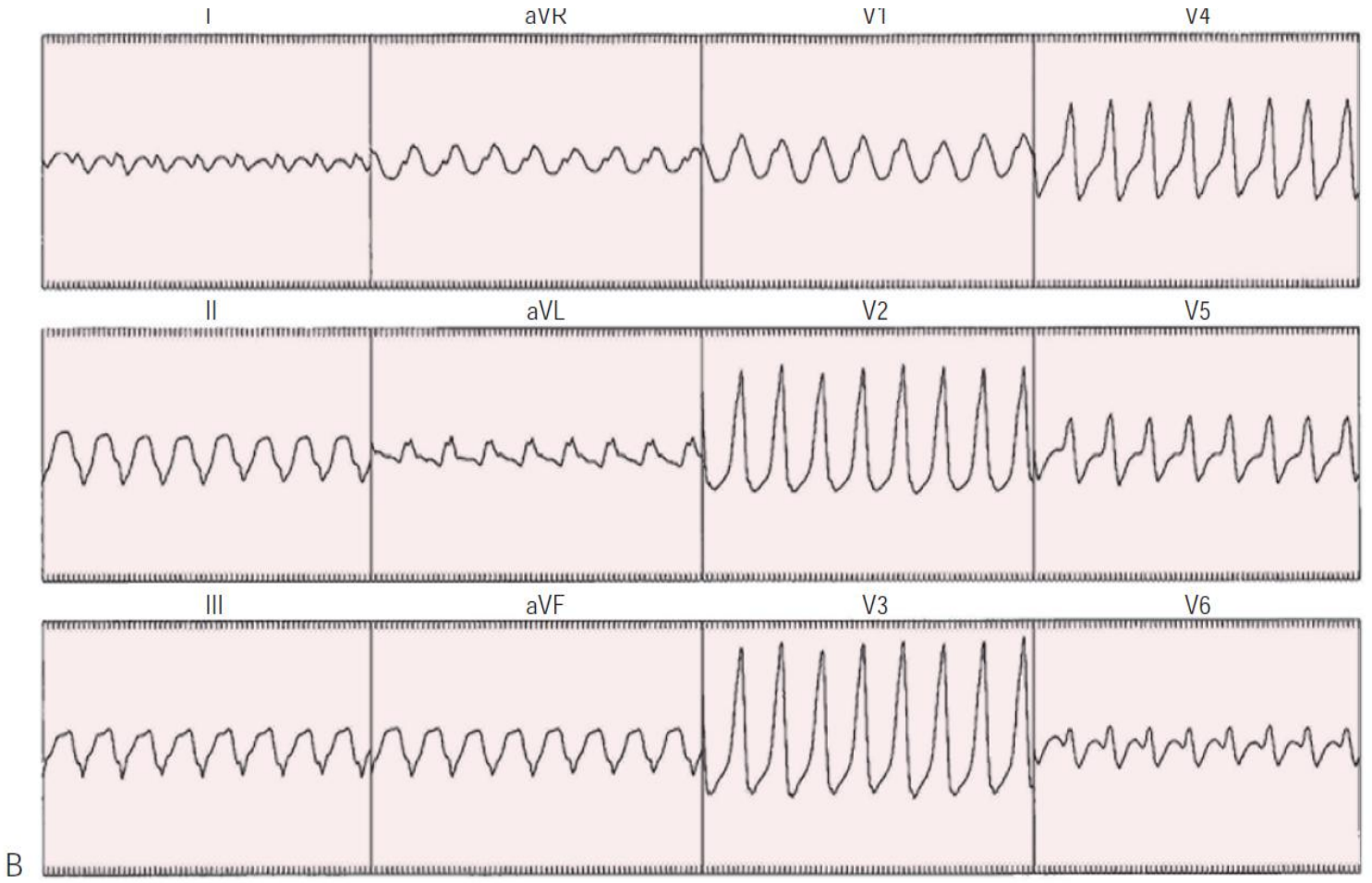
FUSION AND CAPTURE BEATS DURING VT



VT IN A PATIENT WITH A PREVIOUS MYOCARDIAL INFARCTION. THE VT EXIT IS IN THE LEFT VENTRICULAR SEPTUM (LEFT BUNDLE BRANCH BLOCK MORPHOLOGY), INFERIORLY (QS IN II, III, AND AVF) CLOSE TO THE APEX (QS IN V6).



EPICARDIAL VENTRICULAR TACHYCARDIA IN A PATIENT WITH CHAGAS DISEASE. THE VT HAS SHORTEST INTRINSICOID DEFLECTION, GREATER THAN 55% OF THE QRS IN THE PRECORDIUM, AND IS THEREFORE OF EPICARDIAL ORIGIN. BECAUSE IT HAS RIGHT BUNDLE BRANCH BLOCK MORPHOLOGY IN V1 AND QS IN LEADS II, III, AND AVF, THE ORIGIN IS IN THE INFERIOR LEFT VENTRICLE.



ISCHEMIC CARDIOMYOPATHY

In the setting of a remote myocardial infarction, the mechanism of VT is reentry and involves the infarct scar and in particular the border zone

As a result, the VT in this setting is typically monomorphic.

More than one morphology can be seen because of different exit sites from the same circuit resulting in different activation patterns of the rest of the ventricle or reversal of the direction of reentry using the same circuit

- Polymorphic VT or VF in the setting of ischemic heart disease usually occurs during active ischemia or infarction

TREATMENT

Primary suppression with antiarrhythmic drugs

(e.g., amiodarone), implantation with an ICD, antitachycardia pacing,

and ablation are options.

Surgical endocardial resection of the scarred area is also an effective treatment of refractory VT caused by previous infarction.

For recurrent VT or VT storm refractory to medications or ablation, cardiac sympathetic denervation has been effective in limited studies.

HYPERTROPHIC CARDIOMYOPATHY

The risk for sudden death

in patients with hypertrophic cardiomyopathy is increased by :

1.the presence of syncope,

2.a family history of sudden death in first-degree relatives,

3.septal thickness greater than 3 cm, or

4.the presence of nonsustained VT on 24-hour electrocardiographic Recordings

Amiodarone has been useful in some patients with

mildly symptomatic, nonsustained VT but not in improving survival

Dual-chamber pacing, septal alcohol ablation, and myotomy/ myectomy have been useful in reducing the outflow gradient, but their role in reducing ventricular arrhythmias has not been established.

In patients believed to be at high risk for sudden death or those with sustained VT or frequent nonsustained VT, an ICD may be indicated.

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY.

Right-sided heart failure or asymptomatic RV enlargement can be present

Male patients predominate, and most are usually found to have an abnormal right ventricle on echocardiography, RV angiography, or magnetic resonance imaging, although this abnormality may not be apparent on initial evaluation

Patients with arrhythmogenic RV cardiomyopathy have VT that generally has a left bundle branch block contour (because the tachycardia arises in the right ventricle) and can have several morphologies (including those consistent with outflow tract VT).

Findings on the signal-averaged ECG can be abnormal because of delayed conduction in the right ventricle

Arrhythmogenic RV cardiomyopathy can be an important cause of ventricular arrhythmia in children and young adults with apparently normal hearts, as well as in older patients.

desmosomal mutations present in only approximately 50%of cases

ICDs are generally preferable to pharmacologic approaches because of the progressive

nature of the disease and poor prognosis, particularly if the patients have poorly tolerated VT resulting in syncope or sudden cardiac death. RF catheter ablation can be tried but often requires ablation of multiple morphologies, as well as extensive substrate ablation to eliminate all potential reentrant circuits. Because most of the circuits

and scarring are located on the epicardial surface, epicardial ablation is often required.

ARVC

The initial findings can be subtle and often mimic those of outflow tract VT; they are

manifested only by tachycardia and no symptoms of right-sided heart failure.

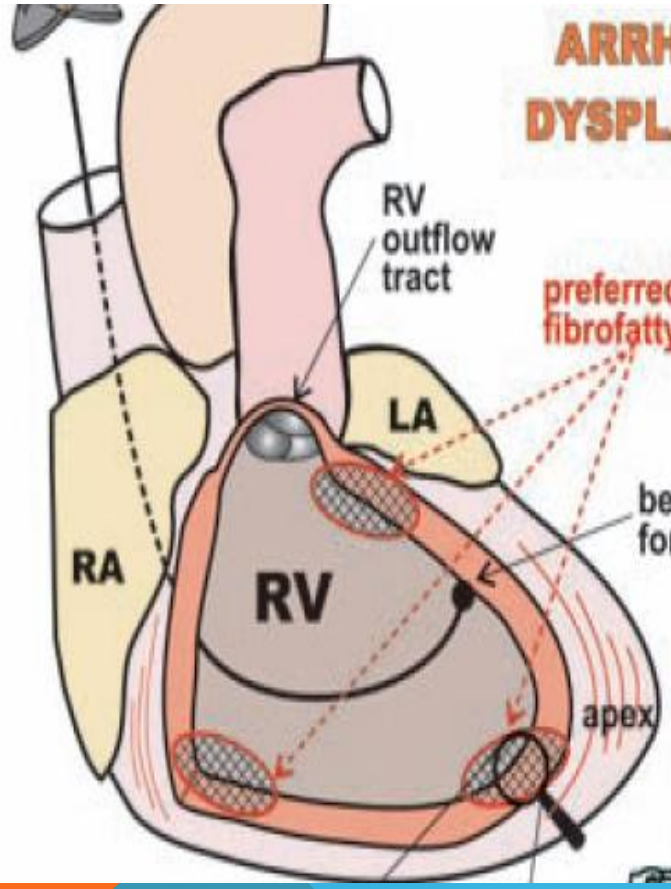
desmosomal mutations present in only approximately 50% of cases, and low penetrance of the inherited trait.

ICDs are generally preferable to pharmacologic approaches because of the progressive nature of the disease and poor prognosis

RF catheter ablation can be tried but often requires ablation of multiple morphologies

Because most of the circuits and scarring are located on the epicardial surface, epicardial ablation is often required.

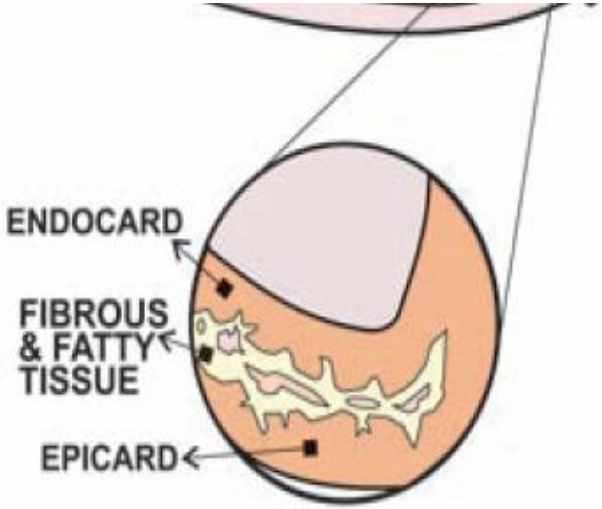
ARRHYTHMOGENIC RIGHT VENTRICLE DYSPLASIA / CARDIOMYOPATHY (ARVD/C)

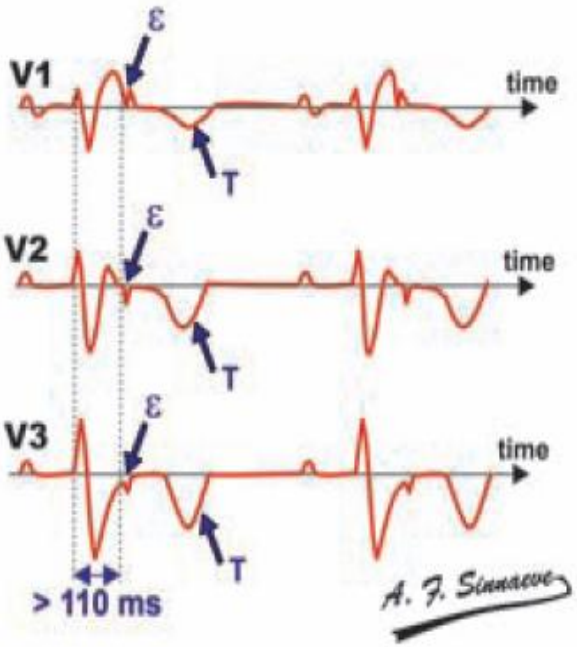


Arrhythmogenic right ventricular dysplasia/cardiomyopathy is a genetic disease characterized by a progressive fibrofatty replacement of the right ventricular myocardium. It is a major cause of sudden cardiac death in young adults in the period between the second and fourth decade of life.

ABNORMALITIES and CLINICAL MANIFESTATIONS

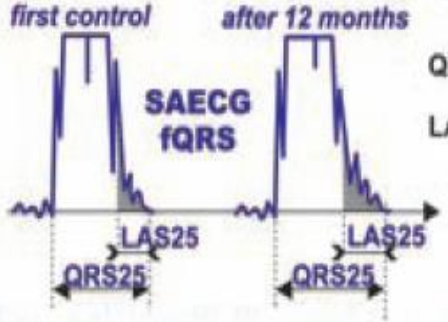
- Depolarization/repolarization abnormalities in the right





Depolarization/repolarization abnormalities in the right precordial ECG leads (V1 - V3).

- ε waves (epsilon waves are small sharp deflections, at the end of the QRS complex, indicating delayed activation of the right ventricle)
- prolongation of QRS duration (any QRS duration > 110 ms may be an indication)
- T-wave inversion (especially in patients older than 12 years of age)
- exercise triggered ventricular dysrhythmias of right ventricular origin with LBBB configuration (monomorphic VT caused by ARVD and degenerating in VF is a well known cause of sudden death in young athletes)
- patients may have RBBB
- late potentials (as detected by signal averaging)



QRS25 : filtered QRS duration with a 25 -250 Hz filter
LAS25 : duration of low amplitude signals (< 40 µV) in the last 40 ms of QRS25

QRS25 ↑ & LAS25 ↑

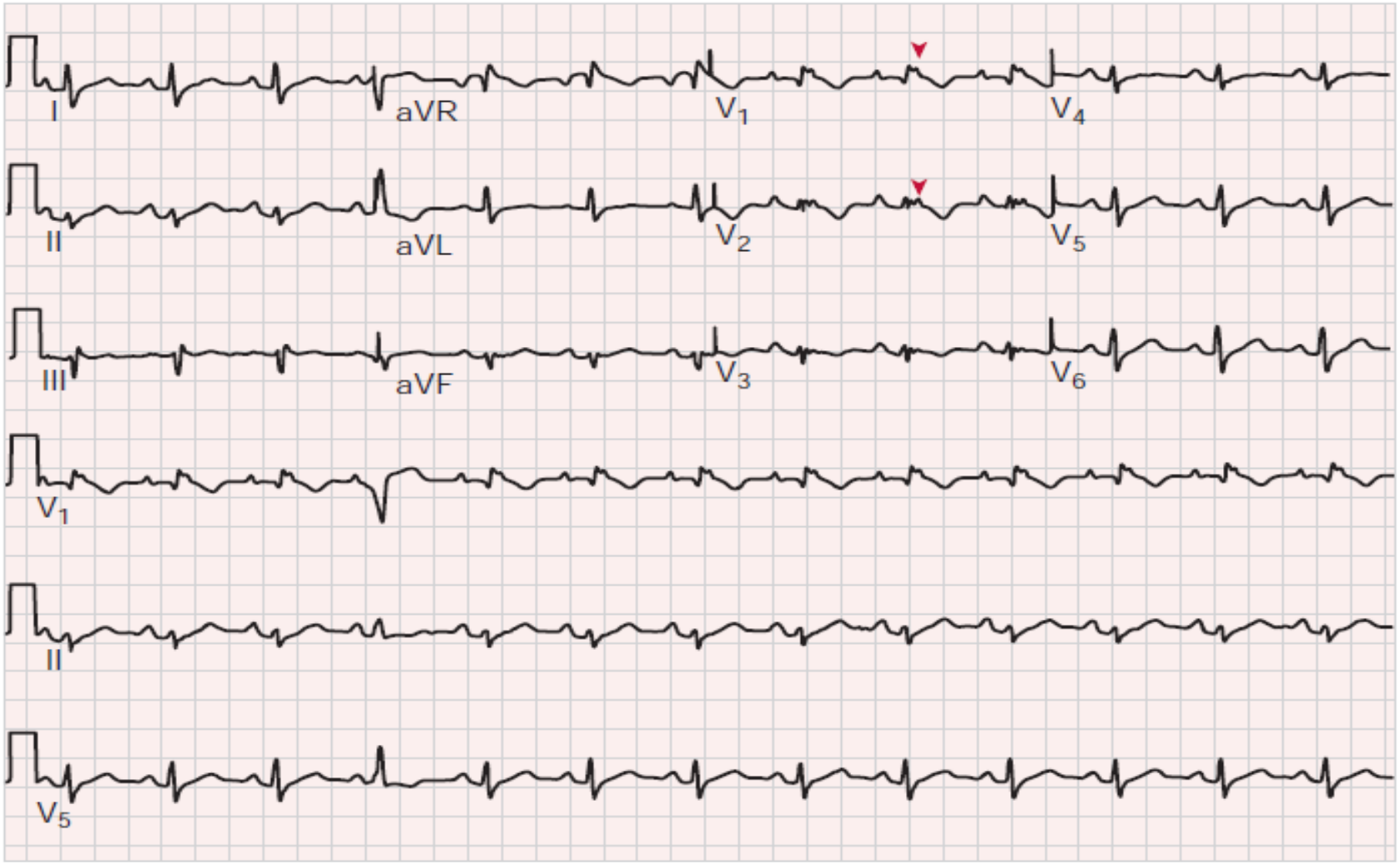
Structural abnormalities of the right ventricle

- the disease is progressive and in rare cases the left ventricle might be affected.

Positive family history of :

- sudden cardiac death, syncope, palpitations, ventricular premature beats, nonsustained or sustained VT.
- There is autosomal dominant transmission with the majority of genetic forms of ARVD/C.

ARVD



ARVD VT

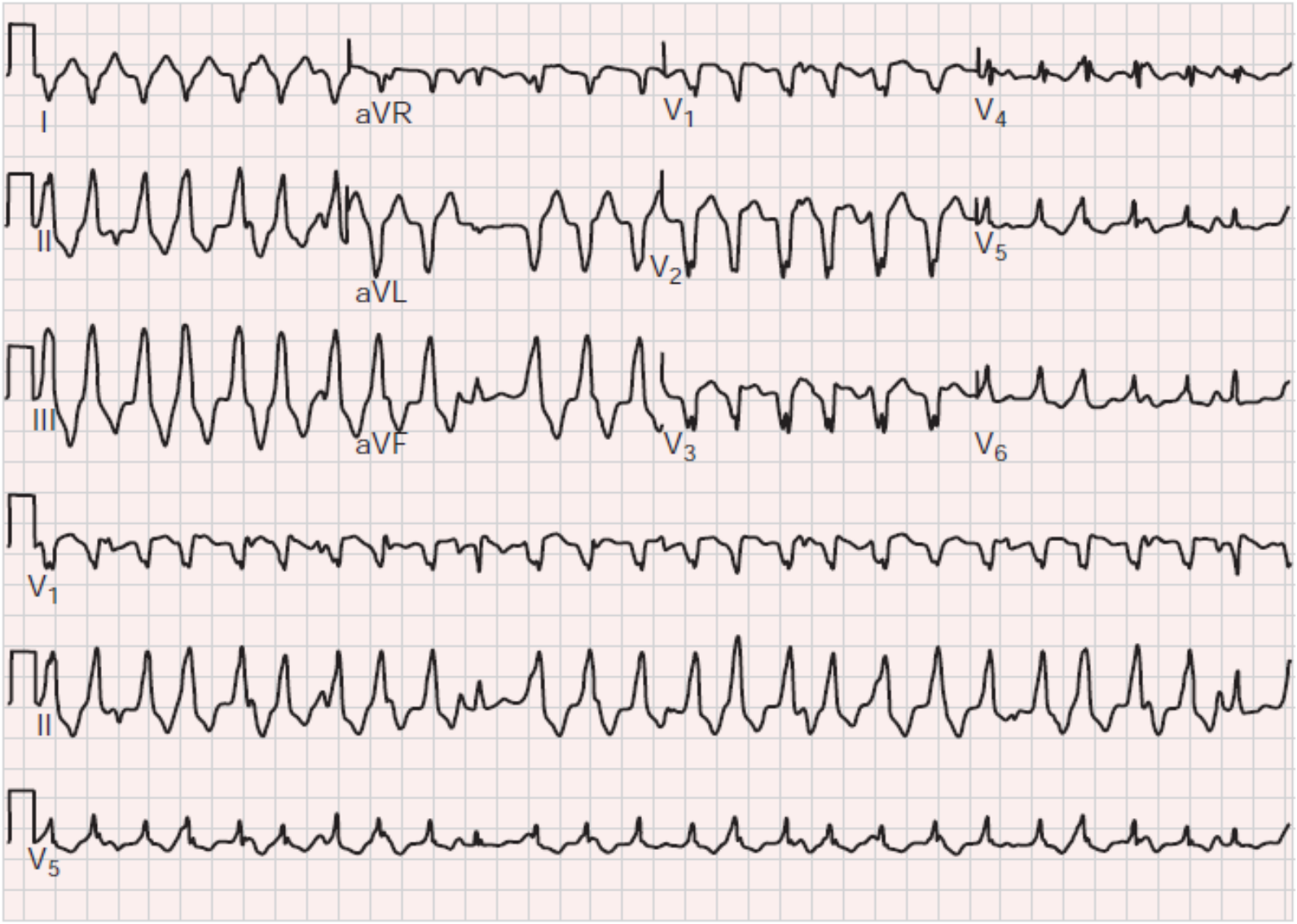


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TABLE 37-9 Diagnostic Criteria for Arrhythmogenic Right Ventricular Cardiomyopathy

Definite diagnosis	2 major criteria *or*
	1 major and 2 minor criteria *or*
	4 minor criteria from different categories
Borderline	1 major and 1 minor criteria *or*
	3 minor criteria from different categories
Possible	1 major *or*
	2 minor criteria from different categories

TETRALOGY OF FALLOT

Chronic serious ventricular arrhythmias can occur in patients some years after repair of tetralogy of Fallot

Findings on the signal-averaged ECG can be abnormal.

In some cases, worsening of pulmonary insufficiency and RV dilation can trigger theVT.

Replacement of the pulmonic valve and concomitant cryoablation of the outflow tract may be required to eliminate the tachycardia.

INHERITED ARRHYTHMIA SYNDROMES

1.CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA:

Patients typically have syncope or aborted sudden death with highly reproducible, stress-induced VT that is often bidirectional.

These patients have no structural heart disease and normal QT intervals

A family history of sudden death or stress-induced syncope is present in approximately 30% of cases

During exercise, typical responses include initial sinus tachycardia and ventricular

extrasystoles, followed by salvoes of monomorphic or bidirectional VT, which eventually lead to polymorphic VT as exercise continues

The treatment of choice is beta blockers and an ICD

Sympathectomy has been reported to be effective in a few cases.

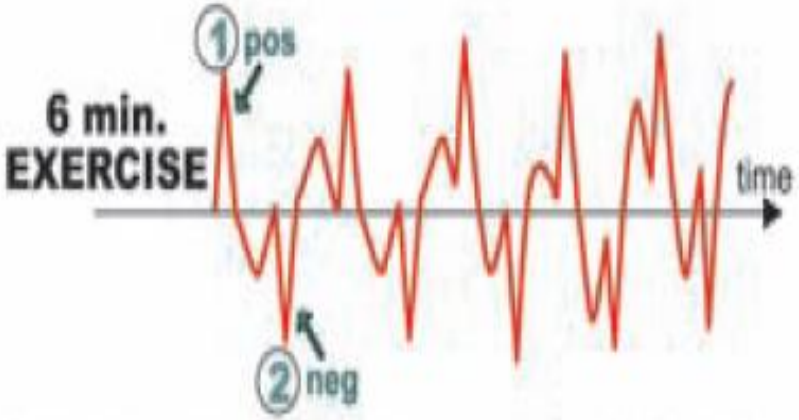
Some distinguishing features of CPVT have been pointed out :

1. A direct relation between adrenergic activation (physical or emotional stress) and the onset of arrhythmias.

2. Absence of structural cardiac abnormalities.

3. An unremarkable resting ECG (with the exception of sinus bradycardia and the presence of "U" waves in some patients).

4. A typical pattern of "bidirectional" VT with an alternating 180° QRS-axis on a beat-to-beat basis
(or an irregular polymorphic VT without QRS vector alternans in some patients).



INHERITED ARRHYTHMIA SYNDROMES

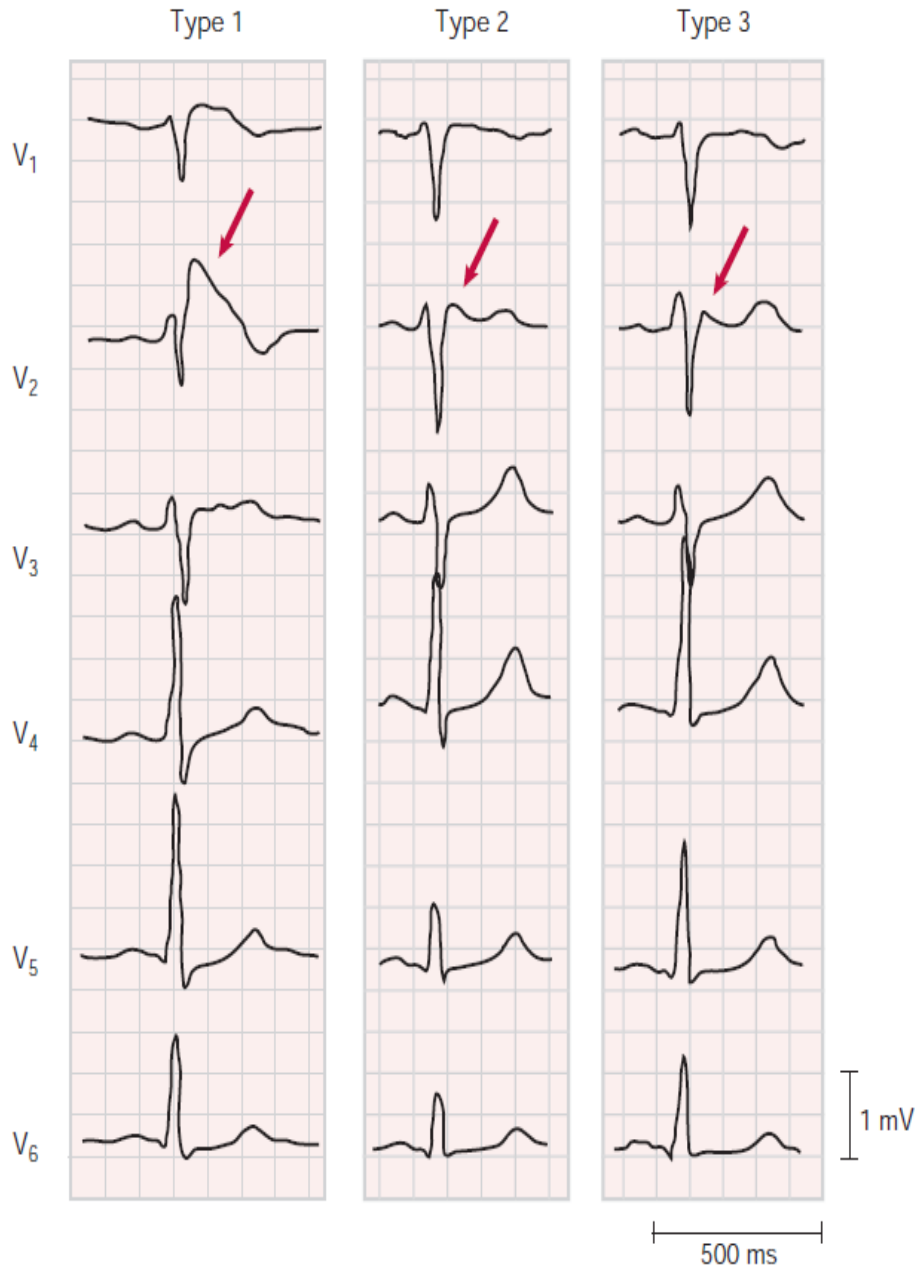
2.BRUGADA SYNDROME

Brugada syndrome is a distinct form of idiopathic VF in which patients have right bundle branch block and ST-segment elevation in the anterior precordial leads, without any evidence of structural heart disease

Findings on the ECG are characterized as type 1, type 2, or type 3 patterns

Brugada syndrome should be suspected in patients with a type 1 ECG pattern in more than one right precordial lead (V1 to V3) if there is documented VF, polymorphic VT, family history of sudden cardiac death, Brugada-pattern ECG in other family members, or syncope

Type 2 and type 3 findings on the ECG are not diagnostic of Brugada syndrome


Type 1
Type 2
Type 3
V1
V2
V3
V4
V5
V6
1 mV
500 ms

If type 2 or type 3 ECG patterns (in more than one right precordial lead) convert to a type 1 pattern after challenge with procainamide, one should consider the diagnosisof Brugada syndrome if at least one clinical criterion is also present

Mutations in genes responsible for the sodium channel *(SCN5A) and calcium* channel have been identified in many families with Brugada syndrome

The precise mechanism of the changes on the ECG and the development of VF is not known. Heterogeneous loss of the action potential dome in the RVepicardium leads to propagation of the dome from sites where it is maintained to sites where it is lost (phase 2 reentry), thereby resulting in ventricular arrhythmias.

Quinidine has been shown to “normalize” the ECG in patients with Brugada syndrome, presumably by blocking the calcium-independent transient outward potassium current (Ito), heterogeneity of which may play a pathogenic role

ablation in the epicardium of the anterior RV outflow tract has also been demonstrated to normalize the ECG, perhaps because of elimination of the Ito-rich area of the RV outflow tract

ICDs are the only effective treatment to prevent sudden death

In patients with VT storm secondary to Brugada syndrome, low-dose isoproterenol

EP studies as a means to risk-stratify patients remains controversial, and a recent study suggested that VT/VF inducibility is unable to identify high-risk patients with Brugada syndrome

the best predictors of a high-risk group:

a.A spontaneous ECG pattern of Brugada syndrome (type 1),

b. a history of syncope,

c. Ventricular refractoriness of less than 200 milliseconds, and

d. QRS fragmentation

TORSADES DE POINTES

VT characterized by QRS complexes of changing amplitude that appear to twist around the isoelectric line and occur at rates of 200 to 250/min

Originally described in the setting of bradycardia caused by complete heart block

The abnormal repolarization need not be present or at least prominent in all beats, but it may be apparent only on the beat before the onset of torsades de pointes (i.e., after a PVC). Longshort R-R cycle sequences commonly precede the onset of torsades de pointes from acquired causes.

VT that is similar morphologically to torsades de pointes and occurs

in patients without QT prolongation, whether spontaneous or electrically

induced, should generally be classified as polymorphic VT, not as torsades de pointes.

The EP mechanisms responsible for torsades de pointes are not completely understood. Most data suggest that early afterdepolarizations are responsible for both long-QT syndrome and torsades de pointes, or at least its initiation.

the most common causes are:

A. congenital severe bradycardia,

B.potassium depletion,

C. use of QT-prolonging medications (such as class IA or III antiarrhythmic drugs). More than 50 drugs have been noted to prolong the QT interval

Women, perhaps because of a longer QT interval, are at greater risk than men for torsades de pointes.

MANAGEMENT

Intravenous magnesium is the initial treatment of choice for torsades de pointes from an acquired cause, followed by temporary ventricular or atrial pacing. Isoproterenol, given cautiously because it may exacerbate the arrhythmia, can be used to increase the rate until pacing is instituted. Lidocaine, mexiletine, or phenytoin can be tried.

When the QT interval is normal, polymorphic VT resembling torsades de pointes is diagnosed, and standard antiarrhythmic drugs can be prescribed

Torsades de pointes resulting from congenital long-QT syndrome is treated with beta blockade, pacing, and ICDs

LONG-QT SYNDROME

The normal QTc interval may actually be 0.46 second in men and 0.47 second

in women, with a normal range of _+ 15% of the mean value

The nature of the U wave abnormality and its relationship to long-QT

syndrome are not clear. The probable risk for development of lifethreatening

ventricular arrhythmias in patients with idiopathic long-QT syndrome is related to the length of the QTc interval, with risk increasing at values of 500 milliseconds or longer.

Long-QT syndrome can be divided into congenital and acquired forms

The congenital form is a familial disorder that can be associated with sensorineural deafness (Jervell and Lange-Nielsen syndrome, autosomal recessive) or normal hearing (Romano-Ward syndrome, autosomal dominant).

Congenital long-QT syndrome is caused by inherited channelopathies created

by mutations in one or more genes

Patients with congenital long-QT syndrome can initially have

syncope, as a result of torsades de pointes

Stress testing can prolong the QT interval and produce T wave alternans, the latter indicative of electrical instability

EP studies are not usually helpful in making the diagnosis.

MANAGEMENT:

In asymptomatic patients with complex ventricular arrhythmias, a family history of early sudden cardiac death, or a QTc interval of 500 milliseconds or longer, beta adrenoceptor blockers such as nadolol at maximally tolerated doses are recommende

Implantation of a permanent pacemaker to prevent the bradycardia or pauses that

may predispose to the development of torsades de pointes may be indicated. In patients with syncope or aborted sudden death, an ICD is warranted. These patients should also be treated with concomitant beta blockers.

An ICD is beneficial in these patients, not simply because of its shocking capabilities but also because of the ability to pace continually for prevention of bradycardia-induced torsadesand algorithms to prevent post-PVC pauses

Use of an ICD in patients without syncope but with a long QT interval and a strong

family history of sudden death is still controversial but may be warranted in selected high-risk patients